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Cleveland Clinic Quarterly

RUSSELL L. HADEN, M.D.

Chief, Department of Medicine, 1930-1948
Consultant in Medicine and Research, 1949-

Born May 22, 1888
Died April 26, 1952

RUSSELL LANDRAM HADEN

RUSSELL HADEN died from a cerebral hemorrhage in the Hospital of the Cleveland Clinic, April 26, 1952, at the age of 63. He died as he had wished, at the height of his intellectual power and surrounded by his family and close friends.

A recounting of his many hours and achievements would hold little interest for him. It is rather by paying tribute to him as a man that we aid in perpetuating those qualities of greatness which characterized Dr. Haden.

Foremost, he was a kind man; kind in his thinking and kind in his actions. His second most prominent quality was the exuberance which underlay his alert and lifelong concern with the phenomena of nature. These qualities he manifested in his study, teaching and practice, in his keen historic sense, his interest in microscopy and his love for his birth state, Virginia. A hater of sham, he lashed out at it no matter whose armor he pierced. Because he believed man was here to work and to work hard, he had no patience with laziness. All this, together with his contagious enthusiasm for the good, made Russell Haden beloved as clinician and teacher, and respected as student of medicine.

He retired at the age of 60 at the full peak of his ability with the aim of doing even more of the things he wanted to do. Immediately, he undertook a program of national service which spanned the world. This and the home at Crozet, Virginia which he and Mrs. Haden had built, he enjoyed to the full. As so often happens, he was not given to live out the span in all its measure. But he was one of the few who could die with a consciousness of fulfillment and free of regret.

His ashes are buried in a surpassingly beautiful spot near the grave of his admired Thomas Jefferson. But his spirit will have approached the new adventure with the same magnificent enthusiasm which added so much to his life on earth.

I. H. P.

SURGERY OF THE AUTONOMIC NERVOUS SYSTEM

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WALTER CANNON,[1] in 1929, coined the term "homeostasis" to describe the autonomic mechanism by which the fluid matrix of the mammalian body is maintained in a constant state despite changes in external environment. This is accomplished by the balanced opposition of the sympathetic and parasympathetic divisions of the autonomic nervous system. The sympathetic division provides an emergency protective mechanism which is always ready to go into action to combat any variety of adverse circumstances. When called into action, the sympathetic division uses up the bodily reserves in order to give rise to an increased liberation of body energy and the effects, therefore, are catabolic in character. Some of the more common conditions which arouse it to activity are pain, hemorrhage, infection, asphyxia, extremes in temperature, and any form of intense emotion. The catabolic functions of the sympathetic system are balanced by the anabolic functions of the craniosacral or parasympathetic division which come into ascendancy during periods of rest and recuperation, and are of a conservative character. When one of these systems is excited, the other is inhibited.

However, this normally efficient homeostatic balance may break down occasionally, resulting in pathologic states. When this occurs the surgeon may be called upon to alleviate the situation by interrupting the flow of impulses responsible for the abnormal state. A list of such pathologic conditions follows:

1. Arterial wounds
2. Arterial embolus
3. Arterial thrombosis
4. Arteriosclerosis obliterans
5. Thromboangiitis obliterans
6. Thrombophlebitis
7. Raynaud's disease
8. Hypertension
9. Hyperidrosis
10. Causalgia
11. Peptic ulcer
12. Carotid sinus syndrome

It is apparent that all but two or three of these pathologic states have to do with a disturbance in the circulatory system. This is not surprising inasmuch as maintenance of circulation is the most obvious function of the autonomic nervous system. But what is the explanation for the disturbed physiology which accompanies these pathologic states? I believe that the answers can be found by studying the normal physiologic response to bleeding.

When an experimental animal is bled by means of a cannula placed in an

artery, there results a progressive reduction in the volume of the circulating blood. The blood pressure, however, does not fall immediately as one would expect, but is maintained at its original pressure by a contraction of the vascular bed. This contraction is occasioned by an increased rate of discharge of vasoconstrictor impulses over the sympathetic division. After perhaps 40 per cent of the animal's blood has escaped, the capacity of this sympathetic vasoconstrictor mechanism is exceeded, and a fall in blood pressure occurs. About this time, however, a vasoconstrictor substance appears in the circulating blood.[2] Thus a humoral mechanism comes to the aid of the overtaxed sympathetic division. By enhancing its vasoconstrictor effect, the fall in blood pressure is combated.

In order to reduce the rate of blood loss, nature has arranged that this vasoconstriction which accompanies blood loss is more pronounced in the wounded limb and especially so in the bleeding vessel itself. The vasoconstriction in the involved limb may be so great as to suggest that nature is willing to sacrifice the injured extremity in an effort to preserve the life of the organism.

This vasoconstrictor response to bleeding has been developed by nature because the chief threat to life in animals in the wild state is wounds of blood vessels. Because this is the case it is not surprising to find that this same phylogenetic response of vasoconstriction occurs as a result of other types of vascular insult.

For example, Villaret and Cachera[3] in 1939 demonstrated that the immediate reaction of the pial vessels to solid emboli was widespread vascular spasm. They found that this spasm was just as pronounced in fields where there were no emboli, indicating its reflex nature. These spastic phenomena were intermittent and constantly recurring, and were seen months after embolization. Spasm and dilatation were found to coexist in parts of the same vessel, and changes in calibre might occur over and over again within a few minutes. The dilated portions of vessels contained cyanotic blood, indicating stasis and hypoxia. They observed that the vessels affected by the disturbance in vasomotricity need not belong to the same parent vessel. They also observed that the venous effects were more widespread than the arterial.

Because of the protective mechanisms with which man has surrounded himself, he has lengthened his life to a point where the so-called degenerative diseases are prevalent. The chief of these is arteriosclerosis. When arteriosclerosis results in the occlusion of one of the major vessels in the lower limb, there occurs this phylogenetic response to vascular insult: i. e. vasoconstriction. The limb, therefore, is deprived not only of the volume of blood which would ordinarily pass through the obstructed vessel, but the volume of blood which passes through the collaterals is reduced by the accompanying vasospasm. The same is true also in varying degree in cases of arterial embolus, thrombosis, thromboangiitis obliterans, and thrombophlebitis.

Kuntz[4] has described the anatomic pathways involved in the neurogenic vasoconstriction which occurs in a limb which is the seat of a vascular lesion. He has demonstrated in the blood vessel walls afferent fibers which transverse

the sympathetic trunk and enter the spinal cord through the dorsal roots of the nerves which convey the corresponding efferent fibers. He states that these afferent nerve fibers which reach the extremity through the sympathetic trunk not only conduct impulses which result in painful sensations, but also impulses which reflexly activate the sympathetic nerves to the extremity. Such reflex stimulation tends to increase vasomotor tonus in the extremity and thereby aggravate the pain. Sympathectomy in the treatment of patients in this category not only interrupts the afferent nerve fibers through which the pain is mediated but also abolishes the reflex vasomotor tonus, thus insuring improvement in the circulation of the limb.

In cases of vascular occlusion it would appear that this reflex arc acts, at times, like a reverberating circuit. The afferent painful impulses initiate vasoconstriction; this in turn, increases the pain which then reflexly aggravates the vasoconstriction.[5] The effects of sympathetic nerve block in various pathologic states may persist far longer than one could explain on the effect of procaine, per se. This is perhaps due to the interruption of this reverberating circuit which then may take many hours or days to return to its former pathologic rate of discharge.

Leriche,[6] as long ago as 1917, showed that when excision of the obstructed segment of an artery is carried out, certain extremities often lose their trophic changes and become more comfortable. This operation of arteriectomy is regaining favor with some surgeons.[7] The improvement in vasoconstriction in the affected limb, which sometimes follows this operation, is undoubtedly due to partial interruption of the reflex arc by elimination of some of the afferent fibers in the blood vessel wall. However, there are relatively few who believe that the results of arteriectomy are as satisfactory as those obtained by paravertebral sympathectomy. When one considers how much more completely the reflex arc is interrupted by paravertebral sympathectomy than by arteriectomy, the reasons for the superior results of sympathectomy become evident.

At the present time nearly all operations on the sympathetic system are directed toward the paravertebral portion of the system which comprises the ganglionated cords. Anterior rhizotomy, periarterial sympathectomy, and division of peripheral nerves, have been largely discarded.

Surgical procedures on the autonomic nervous system advocated at the present time are indicated in the accompanying table.

Sympathetic Block

Cervical sympathetic block appears to be of benefit in certain cases of cerebral embolus, cerebral thrombosis, and cerebral trauma.[8] When one wishes to interrupt the sympathetic outflow to the brain, it can be accomplished satisfactorily by injection well cephalad to the stellate ganglion. Here the injection is far simpler and there is no danger of puncturing the pleura, a risk occasionally encountered with a stellate ganglion block.

A procaine block of the stellate ganglion is chiefly employed in order to

Table

PROCAINE BLOCK	
Cervical sympathetic	—Embolus, thrombosis and trauma of the brain.
Stellate ganglion	—Arterial wounds, embolus, thrombosis, causalgia in upper extremity.
Upper thoracic sympathetic (alc.)	—Angina pectoris.
Splanchnic nerve	—Painful conditions within abdomen.
Lumbar sympathetic	—Arterial wounds, embolus, thrombosis, arteriosclerosis obliterans, Buerger's disease, causalgia, thrombophlebitis in the lower extremity.
Caudal (epidural)	—Same as lumbar.
Spinal (subarachnoid)	—Same as lumbar.
PARAVERTEBRAL SYMPATHECTOMY	
Cervical	—Cerebral vascular disease— (effectiveness still to be proved.)
Upper thoracic	—Arterial wounds, embolus, thrombosis, causalgia, Raynaud's disease, hyperidrosis of the upper extremity, angina pectoris.
Supradiaphragmatic (splanchnicectomy)	—Hypertension. Pain in abdomen. (Peet)
Subdiaphragmatic (splanchnicectomy)	—Hypertension. (Adson)
Thoracolumbar	—Hypertension. (Smithwick)
Complete	—Hypertension. (Grimson)
Lumbar	—Arterial wounds, embolus, thrombosis, causalgia, Buerger's disease, arteriosclerosis obliterans of the lower extremity.
VAGUS NERVE RESECTION	—Peptic ulcer.
GLOSSOPHARYNGEAL NERVE RESECTION	—Carotid sinus syndrome.

determine the degree of improvement which one may anticipate from sympathectomy in conditions of the upper extremity. The usual indications are arterial embolus, thrombosis, and causalgia. Alcohol injection of the upper five thoracic sympathetic ganglia has been advocated by White[9] for the relief of angina pectoris. The number of injections required, and the duration of the postinjection pain in these cases, somewhat discourage the widespread acceptance of this technic.

Procaine injection of the splanchnic nerves causes a temporary interruption of the visceral sensory fibers contained in these nerves. This injection is, therefore, sometimes used in order to determine whether one may anticipate relief of intra-abdominal pain by division of the splanchnic nerves.

Procaine block of the lumbar portion of the paravertebral ganglionic chain is used chiefly as a temporizing measure, and in order to determine the benefits

to be anticipated by lumbar sympathectomy in such conditions in the lower extremities as embolus, thrombosis, arteriosclerosis obliterans, Buerger's disease, causalgia, and thrombophlebitis.

Caudal epidural or the lumbar subarachnoid injection of the anesthetic agent is sometimes used instead of a lumbar sympathetic block. This method of administration of the anesthetic agent, provided that it diffuses far enough cephalad, has the advantage that it produces an interruption of the sympathetic impulses to both lower extremities. However, neither of these latter procedures is as selective as a lumbar sympathetic block, inasmuch as they interrupt all of the sensory nerves from the lower extremities and not merely those which traverse the lumbar sympathetic chain.

Operative Procedures

Excision of the cervical sympathetic chain is being employed in cases of cerebral vascular disease. The effectiveness of this procedure, however, is yet to be proved.

When sympathetic denervation of the upper extremity is indicated because of diminished circulation due to arterial wounds, embolus, or thrombosis, or in cases of causalgia, Raynaud's disease, and hyperidrosis, the procedure most commonly employed is a modification of Smithwick's upper thoracic preganglionic sympathectomy.[10] If sympathectomy is to be performed for the relief of angina pectoris, a more extensive procedure is indicated, such as the excision of the stellate and the four upper thoracic sympathetic ganglia as advocated by Lindgren and Olivecrona.[11]

For the relief of painful conditions within the abdomen, the supradiaphragmatic sympathectomy, sometimes called splanchnicectomy devised by Peet, is preferred.[12] The effectiveness of this procedure in the pain of pancreatitis is described by deTakats.[13] In this case the aim of the surgeon is to interrupt the visceral afferent fibers which traverse the sympathetic system. The interruption of the sympathetic fibers is merely incidental to the accomplishment of this object.

In the treatment of hypertension the four most commonly employed operations are the supradiaphragmatic sympathectomy advocated by Peet, the subdiaphragmatic sympathectomy advocated by Adson, the thoracolumbar sympathectomy advocated by Smithwick[10] and the so-called complete sympathectomy described by Grimson.[14] Considering the morbidity involved in the more extensive procedures, it is my opinion that the supradiaphragmatic sympathectomy of Peet is probably preferable. However, the effectiveness of some of the newer sympathetic blocking agents will probably relegate the surgery of hypertension into discard.

At the present time the most commonly employed operative procedure on the sympathetic nervous system is lumbar sympathectomy. It is indicated in cases of deficient circulation in the lower extremity caused by arterial wounds, embolus, thrombosis, Buerger's disease, arteriosclerosis, and in cases of traumatic causalgia. An occasional case of Raynaud's disease may affect the lower ex-

tremities to the extent that lumbar sympathectomy is indicated. The benefit following lumbar sympathectomy is due not only to the interruption of the efferent stream of sympathetic impulses but also, in part, to the interruption of the afferent painful pulses which traverse the lumbar sympathetic chain and serve to accelerate the rate of discharge of vasoconstrictor impulses. The frequency of vascular disease in the lower as compared with the upper extremities is due to the erect posture which man has assumed and for which nature has not as yet made a satisfactory adjustment.

There are various theories as to why the syndrome of causalgia is frequently relieved by sympathectomy. The explanation I prefer is that offered by Doupe, Cullen, and Chance[15] who believe that afferent discharges over the sympathetic pathways traverse the "artificial synapse" at the site of the nerve injury, and in this way produce impulses in afferent nerves resulting in a sensation of pain. Their work was based on the earlier experiments of Granit, Leksell, and Skoglund,[16] in 1944, who demonstrated by study of the nerve action currents that an artificial synapse is produced in a mixed nerve by injury or pressure. It is, therefore, easy to see how a continuous flow of efferent sympathetic impulses going down a mixed nerve could become short circuited at the point of injury and jump across to the naked axis cylinders of the afferent nerves with the production of a pain impulse.

Operations for the relief of conditions produced by overactivity of the parasympathetic nervous system are vagus nerve resection for peptic ulcer, as advocated by Dragstedt,[17] and resection of the carotid sinus nerve or, more effectively, by resection of the glossopharyngeal nerve intracranially, as advocated by Bronson Ray[18] for the relief of carotid sinus syncope.

Surgery of the sympathetic nervous system has progressed through many phases and it seems likely that the day will come when surgery will no longer be required in the management of these physiologic disturbances.

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TRANSLUMBAR AORTOGRAPHY

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SATISFACTORY visualization of arterial circulation by roentgen examination is a comparatively recent accomplishment. This is true of translumbar aortography, although it was described by Dos Santos before the Surgical Society of Paris in 1929.[1] This diagnostic procedure did not meet with immediate acceptance, however, and almost a quarter of a century elapsed before it became recognized as a valuable diagnostic aid. Improvements in contrast media and technic have contributed to making arteriography a safe and simple examination.[2,3]

The original application of aortography was chiefly in the field of urology to differentiate between various types of renal lesions.[4-8] Useful information concerning potential renal function can be obtained from a roentgenographic study of renal blood supply.

In recent years, aortography has been found to be of equal or even greater value in the diagnosis of vascular pathology involving the abdominal aorta and its immediate branches.[9,10] Suspected conditions such as aneurysm and complete or partial occlusion may now be confirmed or excluded by this means.

The technic of translumbar aortography has been adequately described in the literature. We have used the simple technic of syringe injection employed by Smith et al.[3] The choice of the opaque media, whether it be sodium iodide, neo-iopax or diodrast is largely the preference of the physician making the injection. We have found no appreciable difference in results between 75 per cent neo-iopax and 70 per cent diodrast. Sodium iodide, 80 per cent, will give a contrast shadow somewhat more dense than the other media and may be preferable with a large patient; however, it is more irritating. One point in the technic should be stressed, the importance of making a preliminary film after insertion of the needle and the injection of 10 cc. of 35 per cent diodrast, particularly when there is doubt whether the needle is in the aorta. This insures the proper positioning of the needle and will prevent injection of the concentrated solution into one of the smaller vessels. The preliminary film is immediately processed and viewed and, if the needle tip is in satisfactory position within the lumen of the aorta, the injection of the concentrated solution follows without delay. We believe that thrombosis of one of the smaller arteries may be prevented by means of this preliminary injection. Aortography may be accomplished with or without anesthesia; only morphine premedication and novocain may be necessary in many instances.

Translumbar aortography has been performed approximately one hundred times at the Cleveland Clinic in the past 2 years without serious complication. A few instances of extramural or intramural injection of the aorta have occurred. The dye is absorbed within a few minutes and the only consequence has been mild back pain for one day. The superior mesenteric and splenic artery have been injected accidently without incident.

The needle is usually inserted into the aorta at the level of the interspace between D 12 and L 1 vertebra. This insures filling of the aorta and all its major branches (fig. 1). For special purposes the needle can be inserted at a lower level to avoid dispersion of the dye into the celiac axis. The renal arteries come off at right angles to the aorta usually at the level of the second lumbar vertebra, the left being slightly higher than the right. Before reaching the hilum of the kidney, the renal artery divides into lesser branches which spread out in a fan-like arrangement, extending almost to the periphery of the renal outline.

Anomalous renal arteries may arise separately from the aorta or the proximal portion of the renal artery (figs. 2 and 3). These vessels must be studied carefully to determine that their branches end within the renal shadow. They may cross the ureter in such a way that hydronephrosis is produced. Aortography provides a means of visualizing these aberrant arteries and demonstrates how much of the kidney is supplied by them.

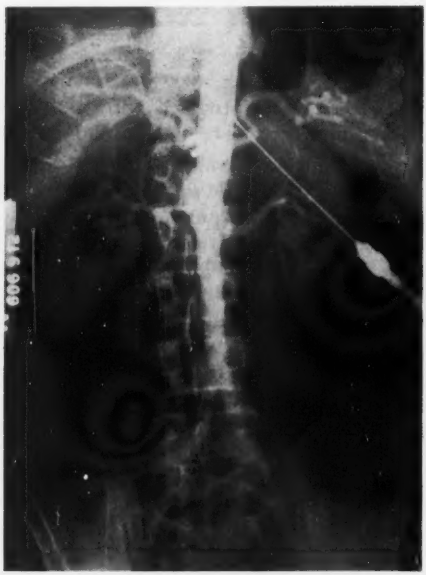

FIG. 1. Normal aortogram. The celiac axis is the first major branch of the abdominal aorta, dividing into the splenic artery, the left gastric and the hepatic artery. The renal arteries arise from the aorta at the level of the second lumbar vertebra.

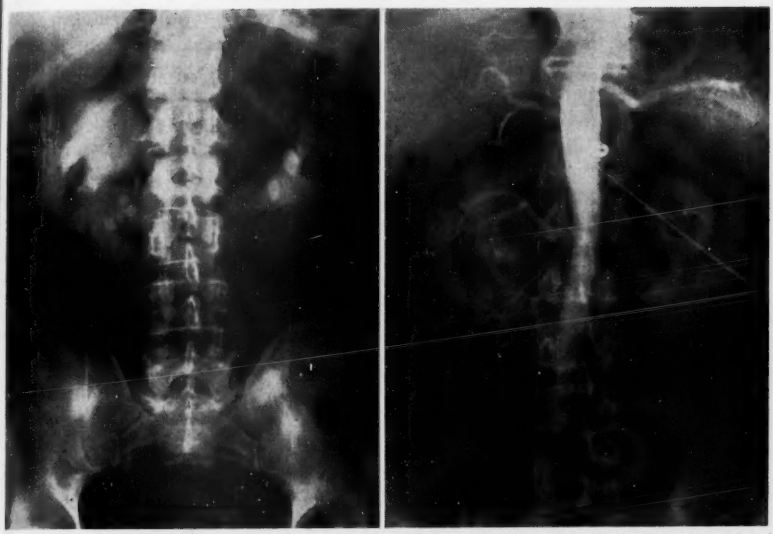

FIG. 2. (a) Intravenous urogram demonstrating a horseshoe kidney with hydronephrosis of right half and multiple calculi in the lower calyces. (b) Two large renal arteries supply the right half of the horseshoe kidney; two large and two smaller arteries supply the left half.

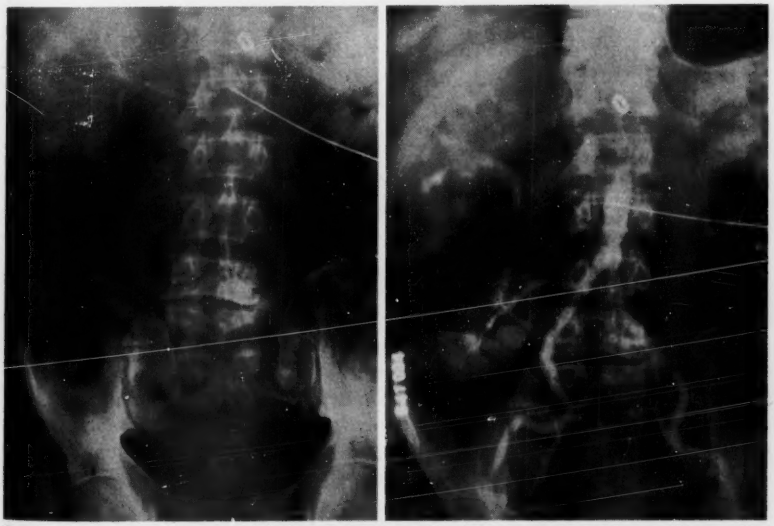

FIG. 3. Crossed renal ectopia. (a) The right kidney lies in its normal position and is supplied by two renal arteries. There is no evidence of a left kidney in its normal location. (b) The second kidney lies on right side of abdomen, deriving its blood supply from right common iliac artery. The opaque medium from first injection is being excreted by both kidneys.

Important information of practical surgical value to the urologist concerning congenital anomalies of the kidneys is supplied by aortography. Horseshoe kidneys have an exceedingly variable blood supply; instances of a solitary renal artery supplying both halves have been observed. The arterial supply of a horseshoe kidney in which two or more renal arteries supplied each half is shown in figure 2. Ectopic kidneys may derive their blood supply from the aorta or the iliac vessels. Figure 3 demonstrates an unusual vascular pattern found when both kidneys are on one side. The first aortogram (fig. 3a) depicts a normal right kidney in its normal position but supplied by two renal arteries. After reinsertion of the needle in the midlumbar aorta, the aortogram was repeated (fig. 3b). The second kidney was found to lie on the right side of the pelvis, supplied by a single renal artery arising from the right common iliac artery. The ureter from the upper kidney can be seen crossing the lower kidney. This condition is known as crossed renal ectopia.

Cysts and malignant tumors of the kidney usually are difficult or impossible to differentiate on pyelographic findings alone. They both cause enlargement and irregularity of the renal outline, and although there often are changes on the pyelogram suggestive of either a cyst or tumor, they may produce an identical deformity of the pelvocalyceal system. Occasionally a cyst or tumor can be present in a kidney without deformity of the renal pelvis or calyces. Under these circumstances, pyelography is of little value. Without exploration or direct needle puncture of the lesion, the two often cannot be distinguished. Aortography has proved useful under such conditions.

A kidney with a cyst has an avascular area corresponding to the pyelographic deformity and enlarged renal outline. There is displacement of the renal parenchyma and intrarenal vessels around the periphery of the cyst. These points are illustrated in figure 4. The pyelogram showed a large, soft tissue mass at the lower pole of the right kidney, with a deformity of the pelvocalyceal system characteristic of either a cyst or tumor. The aortogram (fig. 4a) indicated the area to be avascular, establishing the diagnosis of renal cyst. Attempted aspiration proved unsuccessful because the cyst was multilocular (fig. 4b).

A renal tumor shows increased vascularity with an abnormal reticulated pattern of arterial supply in the region of the pelvocalyceal deformity, known as "pooling" of the opaque media. Some vessels adjacent to the mass may be displaced around it. A small tumor may alter only a portion of the renal blood supply, whereas a large tumor has multiple abnormal vessels and rich vascularity. In figure 5 are shown the roentgenograms of a renal tumor where aortography established the diagnosis preoperatively. The urogram showed a soft tissue mass arising from the upper pole of the left kidney without calyceal deformity. Differentiation between cyst or tumor could not be made. In the aortogram (fig. 5a) a large artery can be seen extending to the center of the mass from which many smaller vessels radiate, indicating that the mass is a tumor (fig. 5b).

A benign adenoma of the renal parenchyma does not show this increased

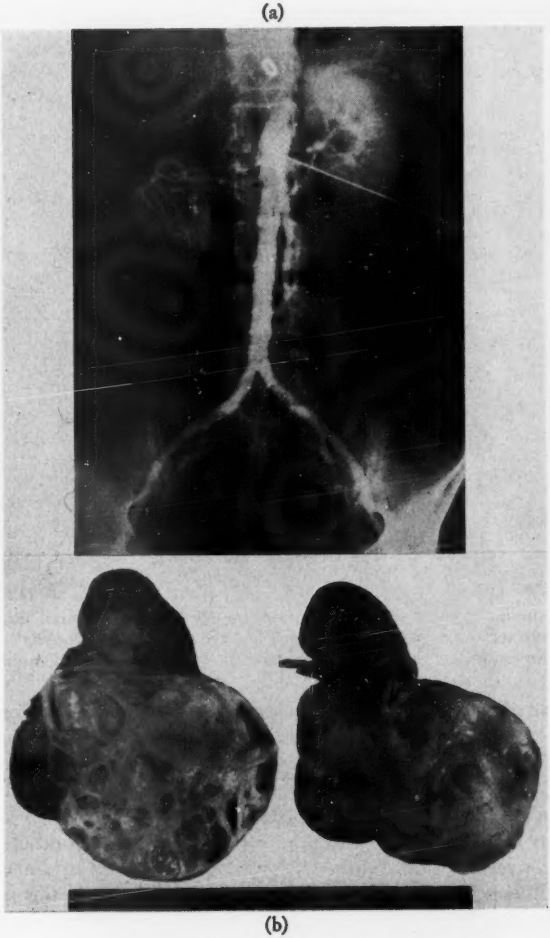

FIG. 4. Renal cyst. (a) Note the abnormal outline of the right kidney with absence of vascularity of the soft tissue mass arising from its midportion. (b) Large multiloculated cyst arising from the midportion of the kidney.

vascularity. Neither do papillary or squamous cell carcinomas of the renal pelvis reveal any characteristic disturbances of the vascular pattern.

Chronic pyelonephritis may cause considerable morphologic change in the kidney. The excretory urogram will show decrease in renal function as well as calyectasis and reduction in renal size. Figure 6 illustrates the renal vascular changes in a patient who had a long history of infection of the left kidney. This

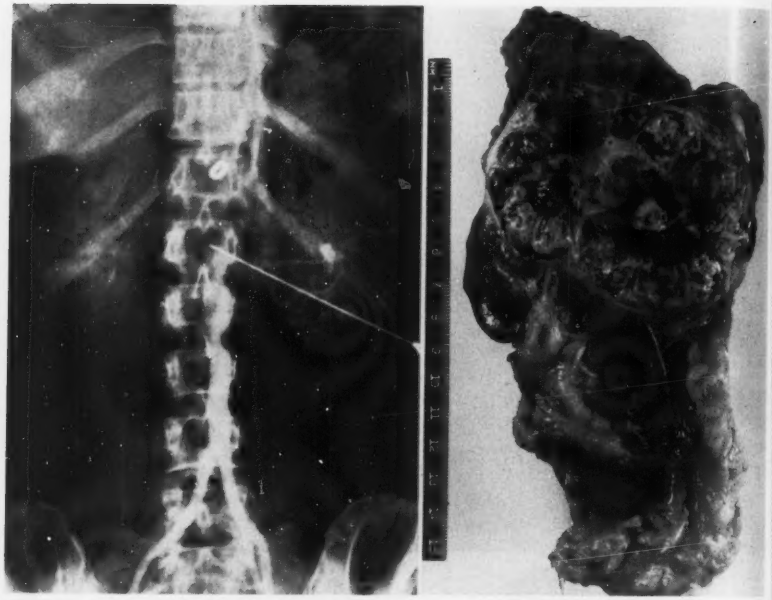

FIG. 5. Renal tumor. (a) The aortogram shows the left kidney displaced downwards by a mass arising from its upper pole. The presence of large vessels and "pooling" of the opaque medium established the diagnosis of renal tumor. (b) Renal cell carcinoma.

kidney was smaller than the opposite one and showed calyectasis on retrograde pyelography. The right renal artery measured 6 mm. in diameter, the left only 3 mm. There was a corresponding reduction in the vascular supply of the entire kidney suggestive of an old inflammatory process. The kidney was removed and showed chronic pyelonephritis.

Ordinarily when a poorly or nonfunctioning kidney is found on urography, a retrograde pyelogram establishes the etiology of the disturbance. When the ureter cannot be catheterized, or the dye fails to get past the ureteropelvic junction, aortography becomes especially useful. A careful study of the vascular pattern within the renal outline furnishes important information concerning the diagnosis and the amount of potentially good renal parenchyma present. Hydronephrosis causes the subdivisions of the renal artery to lengthen and become narrow with decrease in the vascularity of the renal parenchyma. A kidney which is a mere shell due to massive hydronephrosis will show only a few small caliber vessels stretched over the entire mass.

The diagnosis of congenital hypoplasia of one kidney may be difficult to establish by conventional means, particularly when the kidney in question fails to excrete sufficient dye to outline the pelvocalyceal system and the ureter is inaccessible to catheterization. In congenital hypoplasia the aortogram will show only a small renal artery and relative avascularity of the kidney.

Chronic renal infection with or without obstruction, will produce pyelographic changes resembling those of congenital hypoplasia. Such atrophy of the kidney can be further assessed by aortography. In the instance shown in figure 7, atrophy followed an old surgical injury of the left ureter. The kidney had virtually no function on urography and the left ureter could not be catheterized. Only a small renal artery is visible in the left renal area (fig. 7a), the kidney apparently undergoing atrophy from infection and obstruction (fig. 7b).

Retroperitoneal tumors are usually diagnosed by the finding of a ballotable mass on physical examination which causes displacement of the abdominal organs and the kidney or ureter. When complete gastrointestinal and urinary tract investigations fail to disclose the origin of a palpable abdominal mass, aortography becomes exceedingly useful. Careful study of the vascular supply of such tumors will reveal their etiology and aid in surgical intervention.

A large right adrenal carcinoma which caused downward displacement of the right kidney is shown in figure 8. The origin of the adrenal artery and the tumor's rich vascularity is well demonstrated. Aortography has not proved helpful in our experience in attempting to locate small adrenal tumors or to demonstrate adrenal hyperplasia.

The aortogram of a patient who had a left upper quadrant mass is shown in figure 9a. Complete gastrointestinal roentgenograms failed to disclose the

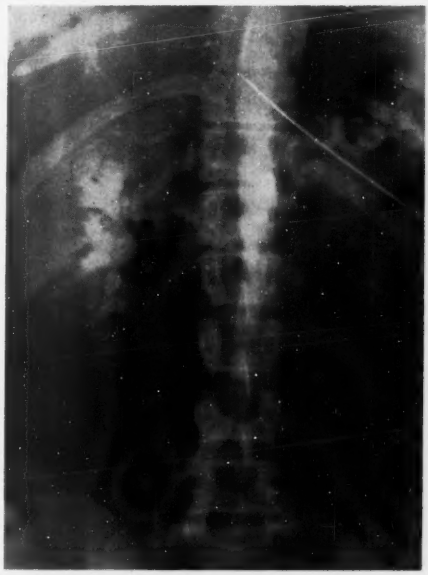

Fig. 6. Combined excretory urogram and aortogram in patient with chronic pyelonephritis of left kidney. Note the difference in size of the two kidneys, the small caliber of the left renal artery and the decreased function of the left kidney.

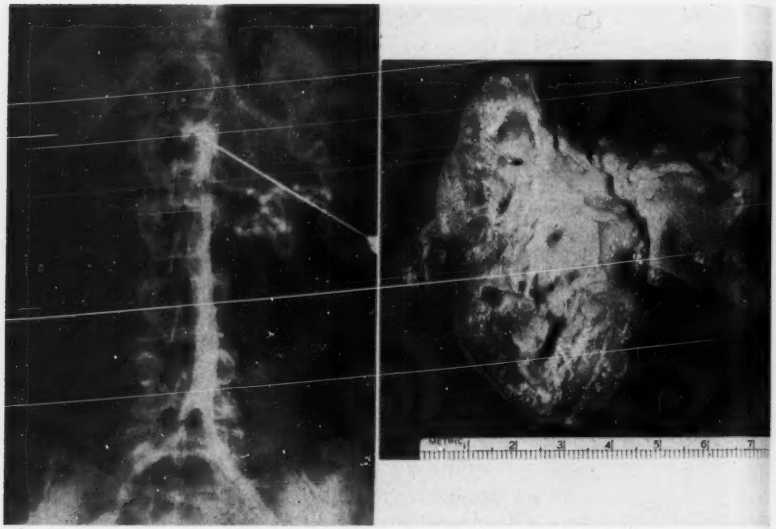

FIG. 7. Atrophy of the left kidney. (a) Note the large right kidney with normal vascularity. Small left renal artery lies below the coiled splenic artery. (b) Small, scarred left kidney.

origin of the mass and it was considered to be spleen, enlarged kidney or a retroperitoneal tumor. Visualization by urography of the left kidney was unsatisfactory and the ureter could not be catheterized for a retrograde pyelogram. An unusually large splenic artery whose branches extended over the entire mass is demonstrated; the kidney is depressed but its blood supply is normal. Hodgkin's sarcoma of the spleen was found at operation, the spleen weighing 1570 Gm. (fig. 9b).

Aortography has shown that occlusive disease of the aorta is more common than previously suspected.[9,10] The aorta may be constricted with only a narrow passageway for blood flow or blocked completely (fig. 10a). The constriction is caused by encroachment of atherosclerotic plaques on the lumen of the aorta. When the plaques ulcerate thrombosis may occur, giving rise to complete thrombotic occlusion of the aorta. The level of occlusion may be anywhere in the lumbar aorta below the renal and superior mesenteric arteries. We have not had an opportunity to observe constriction or occlusion of the aorta above this level except in one boy with severe hypertension due to coarctation of the abdominal aorta commencing just below the origin of the celiac axis.

Patients who have claudication of the low back, hip, and upper leg with an absent or weak femoral pulse in the affected extremity, may be shown to have constriction or occlusion of the corresponding common iliac artery by aortography (fig. 10b). The level of occlusion may be at or close to the bifurcation

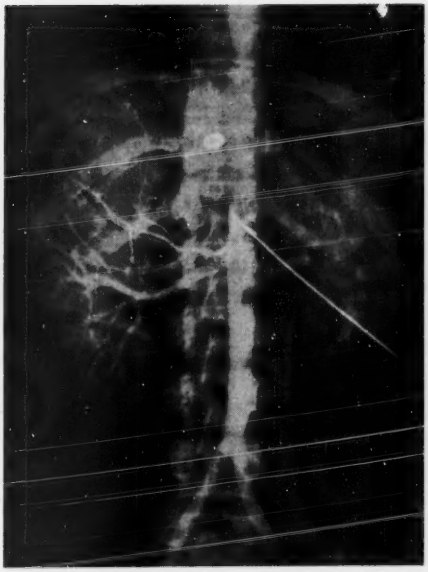

FIG. 8. Carcinoma of right adrenal gland. The right adrenal artery is enlarged, supplying a large mass which displaces the right kidney. Pulmonary metastases are present.

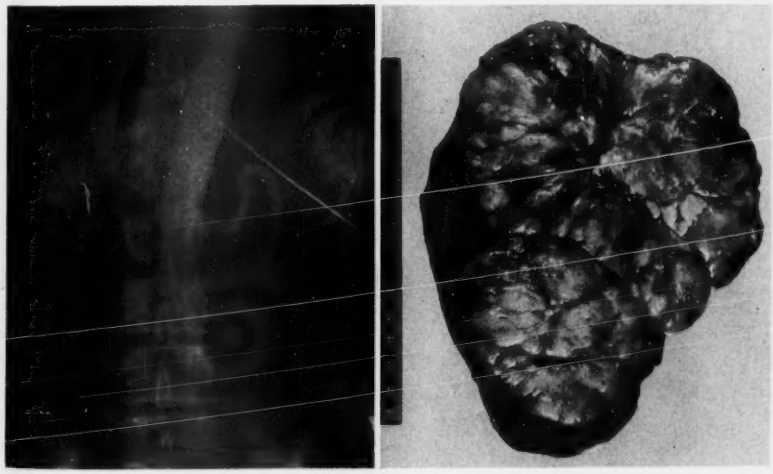

FIG. 9. (a) The left kidney is displaced down to the iliac crest by an enlarged spleen. (b) Hodgkin's sarcoma of the spleen.

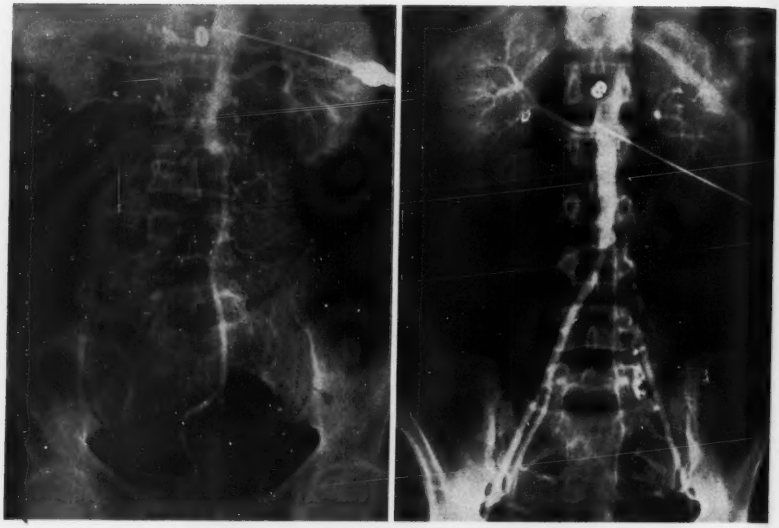

FIG. 10. (a) Occlusion of the abdominal aorta just below the superior mesenteric artery. Anastomotic vessels entering the common iliacs are shown. (b) Occlusion of left common iliac artery. Anastomotic vessels between the aorta and the distal iliac artery are shown.

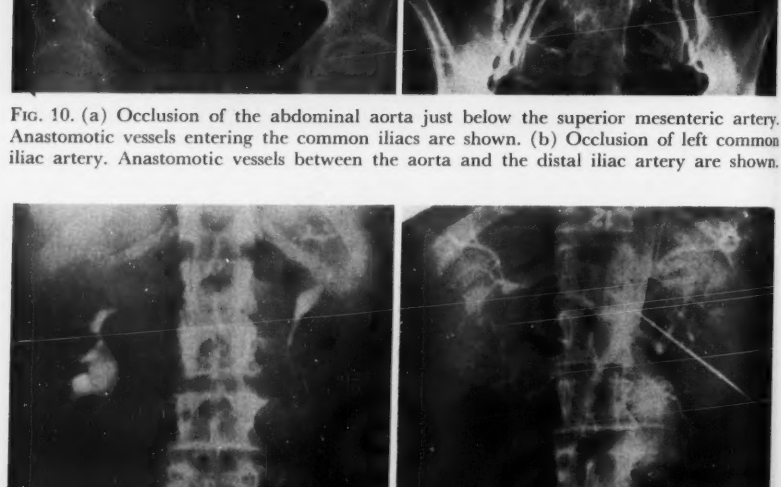

FIG. 11. (a) The left ureter is displaced around a mass. (b) Aortogram demonstrating aneurysmal dilatation of the abdominal aorta.

of the aorta. Anastomotic arteries from the aorta filling the distal portion of the internal and external iliac arteries often are demonstrated.

Aneurysms of the lower thoracic or abdominal aorta, as well as the iliac or femoral arteries, can be visualized by injecting the aorta above the lesion (figs. 11a and b). Direct puncture of the aneurysmal sac has been performed although this is better avoided. The presence as well as exact location of an arterial embolism can be demonstrated by aortography.

Many important and unusual diseases of the aorta and its major branches were seen formerly only at autopsy. Their practical demonstration in patients with vascular disease can now be accomplished by several methods,[2,10,11] each with its own advantages. In our experience, translumbar aortography has proved a reliable and simple means of visualizing the aorta and its major branches.

Summary

Translumbar aortography is a safe, practical and valuable diagnostic aid to the urologist, supplementing intravenous urography and retrograde pyelography in evaluating renal disease. Under certain circumstances which have been described, the demonstration of the blood supply of various organs may be the only way of establishing the diagnosis. Various diseases of the aorta and its major branches can be accurately visualized by translumbar aortography. It is possible that wider applications of this useful procedure will be developed.

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GASTRIC CARCINOMA ASSOCIATED WITH GASTROENTEROSTOMY

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CARCINOMA of the stomach is an uncommon complication in patients who have undergone gastroenterostomy. Gray and Lofgren[1] found only 17 cases of carcinoma in 825 instances where surgical exploration of a previously performed gastroenterostomy was undertaken. The infrequency of coexistent duodenal ulcer and gastric carcinoma has been repeatedly emphasized (Bockus,[2] Eusterman and Balfour,[3] Fischer et al[4]). Orringer,[5] in a recent review of the literature, was able to accumulate only 32 instances of gastric malignancy which developed following surgery for chronic duodenal ulcer.

Gastroenterostomy has been performed in the past as a definitive operation for benign or supposedly benign gastric lesions. One might expect that instances of gastric carcinoma associated with gastroenterostomy would be found in this group. Some of the gastric ulcers thought to be benign at the time of operation might have been, in reality, neoplastic. Furthermore, some authorities believe that a malignant change can occur in a benign gastric ulcer. It is surprising, therefore, to find little in the literature regarding such cases. Gray and Lofgren[1] in their large series, reported only 6 patients with carcinoma of the stomach in whom gastroenterostomy had previously been performed for presumably benign gastric ulcers.

We have recently studied 3 patients in whom cancer of the stomach and gastroenterostomy were found to be associated. We are reporting these cases because of their relative rarity and because they illustrate several interesting points in diagnosis.

Case 1. A 60 year old white male cook was admitted to the Cleveland Clinic November 5, 1951, complaining of anorexia, aerophagia and postprandial distention. These symptoms had begun only a few weeks previously. There was no weight loss, although food intake had been curtailed by postprandial distress.

The past history was significant in that, in 1929, a posterior gastroenterostomy had been performed elsewhere, presumably for a duodenal ulcer. Prior to operation the patient had experienced periodic epigastric distress relieved by food. There had been no recurrence of these symptoms. In 1938 a combined abdominoperineal resection had been performed for adenocarcinoma of the rectum. The lesion was successfully removed despite the fact that there was some extension to the perirectal fat. In 1944 it became necessary to carry out further resection of the sigmoid colon and to establish a new colostomy. No evidence of recurrent carcinoma was found.

Physical examination revealed no significant findings other than a functioning colostomy and a small sac of herniated omentum at the site of the original colostomy.

Laboratory studies showed the erythrocyte count to be 5,580,000 per cu. mm.; the leukocyte count to be 5400 per cu.mm. and the hemoglobin to total 13.5 Gm. per

hundred cc. Gastric analysis, after alcohol stimulation, demonstrated a total acidity of 36 units per hundred cc. with 18 units of free hydrochloric acid. The blood urea was 24 mg. per hundred cc.

A roentgenogram of the chest showed no abnormalities. X-rays of the stomach after a barium meal demonstrated narrowing of the distal two-thirds with polypoid change in the antrum (fig. 1). These changes were thought to represent an infiltrating neoplasm. A barium enema showed no abnormality of the colon.

An exploratory laparotomy was carried out and a large carcinoma of the stomach was found. There was extensive spread of the neoplasm throughout the peritoneal cavity, and ascites was present. The carcinoma was judged inoperable, and no palliative procedure was possible. A biopsy of the omentum showed "carcinoma, simplex, colloid in type." Postoperatively the patient was discharged for terminal care.

Case 2. This patient was first seen at the Clinic in September 1948 for gastroscopic examination only. He gave a history of epigastric distress of many years' duration. Gastroscopic examination revealed a linear ulcer of the lesser curvature surface of the antrum which had the appearance of a benign lesion.

In October 1948 the patient had an operation at another hospital during which posterior gastroenterostomy was performed and bilateral vagotomy done. His condition was apparently satisfactory until the fall of 1950 when postprandial epigastric pain developed which was relieved by antacids. The pain progressed in severity, the patient noted slight weight loss and experienced a general decrease in vitality.

At the time of admission on August 21, 1951, physical examination was normal except for a well healed right upper quadrant incisional scar. Routine urine analysis was essentially negative. There were 3,420,000 erythrocytes and 9850 leukocytes per cu. mm. and 8.8 Gm. of hemoglobin per hundred cc. Gastric analysis, after both alcohol and histamine stimulation, showed no free hydrochloric acid. Blood urea was 30 mg. per cent. Wassermann and Kahn tests were negative.

A roentgenogram of the chest was completely normal. A study of the stomach after a barium meal proved the stoma of the gastroenterostomy to be functioning and demonstrated antral deformity with probable ulceration (fig. 2). A malignant lesion was suspected by the roentgenologist.

Gastroscopic examination revealed narrowing and fixation of the antrum consistent with a diagnosis of infiltrating neoplasm.

Laparotomy was performed and an ulcerating but nonpenetrating carcinoma of the antrum of the stomach was found. This lay along the lesser curvature distal to a patent gastroenterostomy. The proximal duodenum was normal. The neoplasm was successfully resected, together with the lower two-thirds of the stomach, and a gastrojejunal anastomosis made. The resected neoplasm was found on histologic study to be a superficial adenocarcinoma with grade III dedifferentiation. No regional nodes were involved.

The patient made an uneventful recovery, and when last seen on February 21, 1952, appeared well although he complained of symptoms of a mild "dumping syndrome."

Case 3. A 50 year old service station attendant had experienced recurrent epigastric distress for 2½ years prior to admission to the Clinic. At the onset his symptoms consisted of postprandial distress relieved by vomiting. Gastric ulcer was diagnosed elsewhere and, 27 months prior to admission, posterior gastroenterostomy was performed. The patient was well until 12 months prior to entry when gastrointestinal bleeding began, as evidenced by tarry stools. Epigastric pain relieved by vomiting

recurred throughout the ensuing year. Symptoms were partially controlled by medical ulcer management but pain became progressively worse and the patient lost about 35 pounds. There were no pertinent physical abnormalities at the time of admission other than generalized abdominal tenderness.

Routine urine analysis showed no abnormalities. There were 10.8 Gm. per hundred cc. of hemoglobin and 9850 per cu. mm. leukocytes. The Wassermann and Kahn tests were negative. Blood urea was 30 and fasting blood sugar 83 mg. per hundred cc. Gastric analysis, following histamine stimulation, showed a total acidity of 56 units and a free hydrochloric acid of 42 units.

Roentgenograms of the chest and the kidney-ureter-bladder area were noncontributory. The gallbladder was visualized following administration of cholecystographic dye and found to be normal. Study of the stomach revealed a normally functioning gastroenterostomy. A bizarre mucosal pattern was observed in the distal portion of the stomach which was thought to represent carcinoma and the first portion of the duodenum was deformed.

Laparotomy was performed and a large neoplasm of the antral and pyloric areas was found. This was deeply ulcerated and appeared to arise from the posterior wall. The neoplasm had spread beyond the stomach and only a palliative resection was possible. Histologic examination of this tumor revealed scirrhous carcinoma of the stomach with lymph node metastases.

The patient was discharged on the eighth postoperative day.

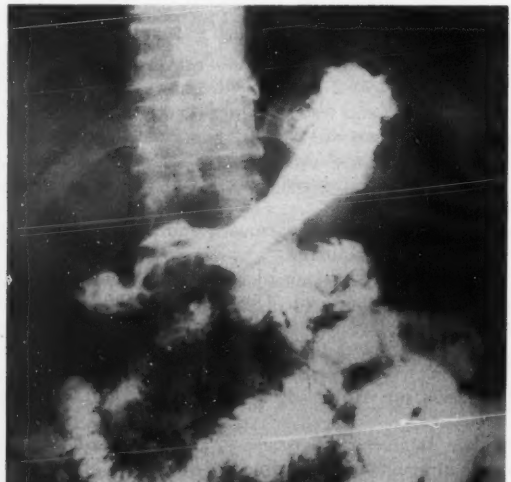

FIG. 1. Case 1. Roentgenogram of stomach. A functioning gastroenterostomy is demonstrated. Distal to this may be seen narrowing of the antrum and a large filling defect. This proved to be an inoperable carcinoma.

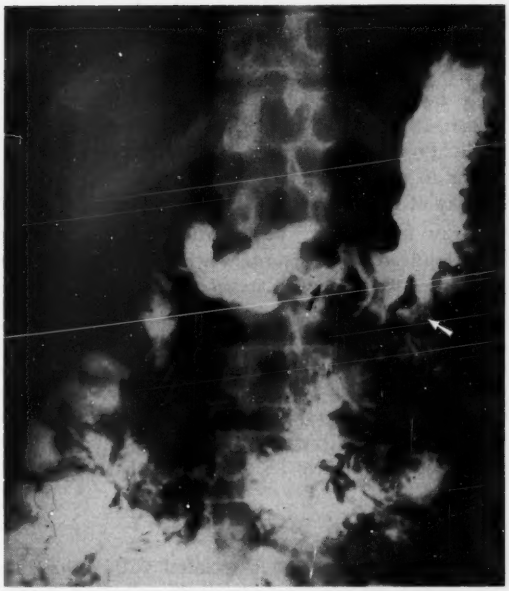

FIG. 2. Case 2. Roentgenogram of stomach. Arrow on right shows site of gastroenterostomy; that on left points to a large ulcerating neoplasm in distal portion of stomach.

Discussion

It is common practice to minimize the possibility of a gastric neoplasm in a patient who has been shown to have a duodenal ulcer. Complications following gastroenterostomy are almost automatically considered to be of a more benign nature. Our first case demonstrates the fallacy of such thinking; here the diagnostic problem was simplified by the fact that the symptoms were of recent origin and did not resemble the typical pattern of peptic ulcer. This patient had no evidence of anemia or achlorhydria, which, had they been present, would have furnished valuable diagnostic clues. Lofgren and Gray[1] found either achlorhydria or a low free acidity in all of their patients who were in this group. X-ray studies of the stomach (fig. 1) were strongly suggestive of an extensive infiltrating neoplasm, and laparotomy confirmed this diagnosis. It is interesting that this patient developed a second malignant neoplasm of the gastrointestinal tract some years after having been cured of the first.

The second and third patients presented a more complicated problem. Here gastroenterostomies were performed for presumably benign gastric ulcers; in retrospect these may well have been malignant lesions. The rather vague and prolonged symptoms recited by the second patient did not suggest malignant disease. The diagnosis was established by a number of objective findings which included moderate anemia, achlorhydria following histamine

stimulation, and roentgenologic demonstration (fig. 2) of an ulcerating lesion in the antrum. Gastroscopic visualization of the antrum provided valuable collateral evidence.

The third case had several features suggestive of benign ulceration. The course was of more than 2 years' duration; the symptoms were periodic and responded to medical management. Gastrointestinal hemorrhage had occurred nearly a year prior to the final laparotomy. Of the objective data available, only the x-ray study of the stomach suggested the presence of neoplasm.

Lofgren and Gray[1] found that 7 of 11 patients who had gastroenterostomies for gastric lesions required further surgery within 4 years and in the majority of instances were proved to have carcinoma. Our second and third cases exhibited a similar course. The short interval between operations strongly suggests that the original lesions were malignant. These cases demonstrate graphically the danger of any operation short of gastric resection for gastric ulcer. All clinical evidence may indicate a benign lesion and the ulcer may appear benign to the surgeon, yet prove subsequently to be malignant. The diagnosis can only be made with certainty by the pathologist. Tissue diagnosis should be obtained of any gastric lesion for which surgery is necessary.

Summary

Three cases of gastric carcinoma associated with gastroenterostomy have been presented and the diagnostic features discussed. Two of these patients illustrate the inadequacy of any procedure other than gastric resection in the surgical treatment of gastric ulcer.

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DISAPPEARANCE OF DIABETES DURING ESTROGEN THERAPY IN ACROMEGALY

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THE development of diabetes in acromegaly is a rather common occurrence. Coggeshall and Root,[1] in reviewing 153 cases of acromegaly, including the 100 formerly reported by Davidoff and Cushing,[2] found glycosuria present in 36 per cent and diabetes in 17 per cent.

Barnes et al,[3] Nelson and Overholser,[4] and more recently Rodriguez,[5] have reported the favorable effect of estrogen administration in experimental pancreatic diabetes in dogs, monkeys, and rats as well as in animals made diabetic

Table 1

ACROMEGALY

Date	Therapy	Hand Volumes	
		R.	L.
8- 6-48	Ethinyl estradiol 1 mg./day for 13 weeks	485 cc.	450 cc.
8-26-48	No therapy for 20 days	500 cc.	485 cc.
9-30-48	No therapy for 53 days	550 cc.	515 cc.
7-28-50	Ethinyl estradiol 1 mg./day 2 mg./day for 52 weeks	525 cc.	438 cc.
11- 9-50	No therapy for 10 weeks	545 cc.	520 cc.

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by crude anterior pituitary extracts. They postulated an estrogen suppression of the pituitary activity concerned with carbohydrate metabolism.

There is some evidence to suggest that estrogens are capable of inhibiting pituitary growth hormone. Thus Zondek,[6] in 1936, produced inhibition of sexual development and dwarfism in the rat and chick by means of estrogenic hormone administration, inhibition which was overcome after administration of Evans' growth hormone. Beneficial clinical results have been reported after the use of estrogens in the treatment of acromegalic patients, by Kirklin and Wilder,[7] Schrire and Sharpey-Schafer,[8] Goldberg and Lisser,[9] Stephens,[10] Reifenstein et al[11] and Kinsell et al.[12] The doses used varied from 1,000 I. U. (as theelin) to 10 mg. per day (estradiol benzoate) parenterally, or up to 5.0 mg. (natural conjugated estrogens) orally. Beneficial results were judged by amelioration in the clinical condition[7-12] as well as a drop in the serum phosphorus and growth hormone levels.[12] These results were variously interpreted as due to inhibition of pituitary growth hormone activity[7,9-12] or to suppression of an abnormality of metabolism associated with the gonadotropic hormones of the overactive pituitary gland.[8]

The observations of Young[13,14] on the production of diabetes in the dog, cat, and ferret by means of a relatively pure growth hormone preparation, suggest that in the acromegalic, diabetes is due to an excess production of this principle. However, since the absolute purity of these preparations is uncertain, other pituitary principles may be involved.

The reversal of diabetes by treatment with large doses of estrogen in one case of acromegaly constitutes the subject of the present report. Other observations on a larger group of patients similarly treated will be reported later.

Methods

Glucose tolerance tests were done using a 100 Gm. single oral dose of dextrose. With this technic the upper limits of normal blood glucose, in our experience, are considered to be as follows: fasting 110 mg., ½ hour 170, 1 hour 170, 2 hours 130 and 4 hours 110 mg. Hand volumes were measured roughly by water displacement, a technic which, though not accurate enough, clearly demonstrated the trends in hand size.

Case Report

A 45 year old woman was referred to the Clinic because of acromegaly, prolapse of the uterus, and an accompanying arterial hypertension. Coarsening of the facial features, enlargement of the hands and feet (figs. 1a and b), and moderate hair growth on the arms and legs had been progressing for at least 8 years. Amenorrhea had occurred abruptly 6 years previously. There were severe headaches daily and 3 weeks prior to her examination she had first been advised of the presence of hypertension. Family and personal histories were noncontributory.

Physical examination revealed the features associated with severe acromegaly, including dorsal kyphosis which prevented the patient from lying flat. There was moderate hirsutism and acne over the face and chin. The thyroid was slightly enlarged

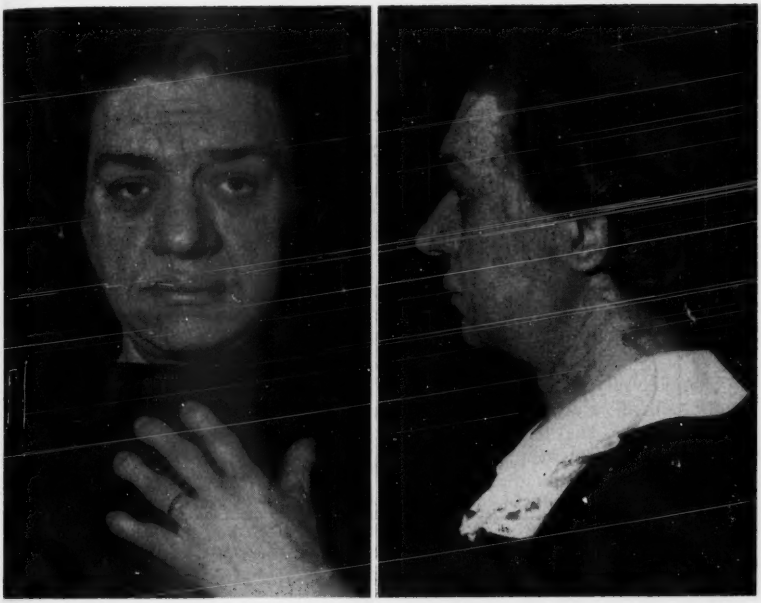


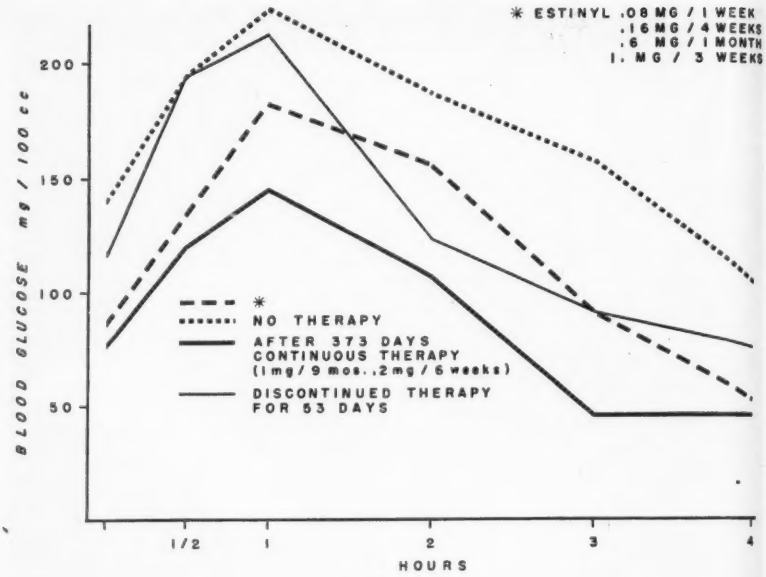
FIG. 1

and nodular. The heart was enlarged to the left. Blood pressure was 194/130. There was a severe uterine prolapse.

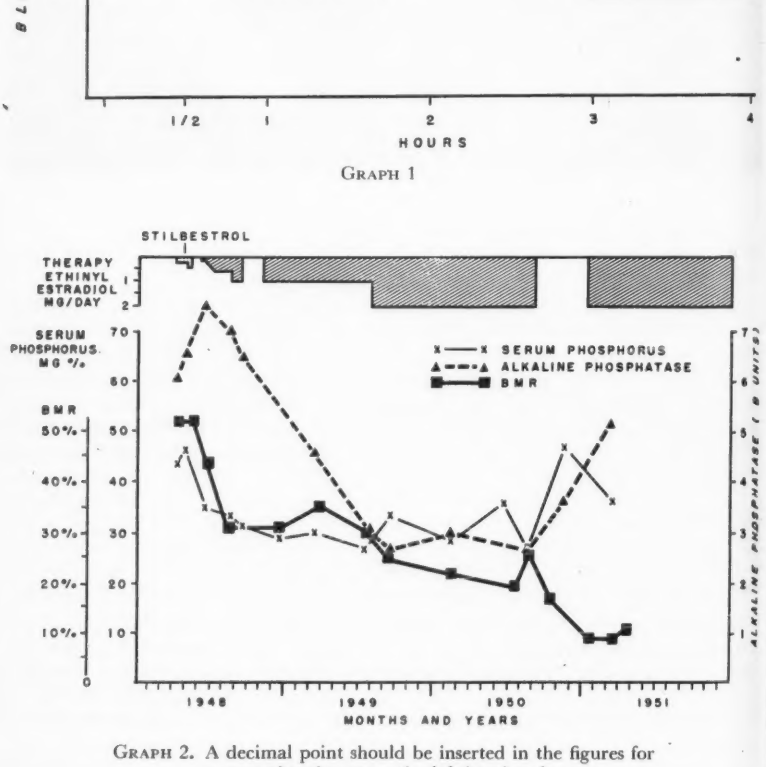
Laboratory examinations were as follows: the urine showed a specific gravity varying from 1.012 to 1.020, with a trace of albumin on several examinations. Hemoglobin, white blood count, blood sugar and blood urea were normal. Wassermann and Kahn reactions were negative. The serum phosphorus was 4.3 mg. per hundred cc., serum calcium 11.5 mg. and the alkaline phosphatase 6.1 Bodansky units. Basal metabolic rate was plus 51 per cent and serum cholesterol 187 mg. per hundred cc. Radioactive iodine uptake was 8 per cent at the end of 2 hours. The glucose tolerance curve was definitely abnormal. Urinary gonadotrophins were less than 13 m.u. and less than 6.6 m.u. per 24 hours, on consecutive examinations. X-ray of the sella turcica showed great enlargement with depression of the floor, and thinning of the dorsum sellae and posterior clinoid processes. There were abnormally prominent frontal sinuses, hyperaeration of other sinuses, and prognathism. X-ray examination of the hands showed tufting of the terminal phalanges. Visual fields for form and color were not diagnostic.

Stilbestrol therapy, 1 mg. daily increasing to 2 mg. daily, was begun but was discontinued after one month because of severe nausea. Ethinyl estradiol* was begun in 0.04 mg. doses per day, increasing gradually to 1 mg. per day, which was continued for 3 months with disappearance of the severe headache and backache, decrease in the coarseness of the features and a diminution in the size of the nose, hands and feet.

Estinyl was supplied through the courtesy of Dr. Edward Henderson and the Schering Corporation.



Graph 1



Graph 2. A decimal point should be inserted in the figures for serum phosphorus on the left hand scale.

Hand volumes after treatment were: left 450 cc., right 485 cc. Therapy was discontinued for 2 months during which time the hand volume increased to: left 515 cc., right 550 cc., along with a return of occipital headache, backache, increase in the size of the nose, lips and tongue.

One milligram of ethinyl estradiol daily was resumed again for a 9 month period after which the dosage was increased to 2 mg. daily for the next year, when treatment was again discontinued. During this period there was complete subsidence of all symptoms and a noticeable increase in strength; her rings became loose, thimbles fit her fingers for the first time since the onset of her illness, and fine movements again became possible. Excessive perspiration ceased. No alteration in blood pressure occurred. Changes in hand volume are shown in table 1.

During the succeeding 4 months the patient was re-examined. Symptoms returned as on the previous cessation of estrogen administration.

Alterations which occurred in the glucose tolerance, serum phosphorus, alkaline phosphatase levels and basal metabolic rate are demonstrated in graphs 1 and 2.

Shortly after the cessation of estrogenic therapy the patient developed a mild cerebrovascular accident from which she gradually recovered. It was at this time that she first noticed blurring of vision, especially laterally, and visual field examinations done repeatedly showed a progressive development of a bitemporal hemianopsia. Because of this finding and the return of active acromegaly ethinyl estradiol, 2 mg. daily was begun again. However, since no immediate improvement in visual fields occurred, the patient received a course of x-ray therapy directed at the pituitary, calculated to deliver approximately 2,000 roentgen units to the gland. This produced no additional effect. There has subsequently been a slow progressive improvement on continued estrogenic therapy and, when last seen after 11 months of continuous treatment, she felt greatly improved, more so than for many years in her own estimation. Arterial hypertension, however, had not diminished. Average blood pressure was 210/120 mm. Hg.

Discussion

We feel that the reversal of the abnormal glucose tolerance test in acromegaly during estrogen administration is further evidence of a potent hormonal inhibition to the pituitary. This is a clinical demonstration of what has been shown previously in experimental animals.[3,6] Whether this improvement in carbohydrate metabolism is due to an inhibition of growth hormone or some other pituitary factor is still a matter of conjecture. To our knowledge this is the first reported instance of complete reversal of diabetes caused by estrogen in the human. Whether or not the pituitary growth hormone or other pituitary factors have a bearing on the etiology of the average case of clinical diabetes remains for future studies to demonstrate.

Summary

A case of acromegaly accompanied by diabetes and arterial hypertension in a 45 year old woman has been presented.

Administration of estrogen was followed by a shift in the glucose tolerance curve from diabetic type to complete normality.

There was also a fall in elevated inorganic serum phosphorus levels to

normal, and considerable clinical improvement including reduction in size of the nose, hands and feet. No effect on the elevated blood pressure was seen.

Discontinuance of estrogen therapy resulted in reappearance of the diabetic type of glucose tolerance.

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TUMORS OF THE PAROTID GLAND

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TUMORS of the parotid gland are of particular interest for several reasons: first, despite years of intensive study since Virchow's first description of the parotid mixed tumor in 1863, both their origin and pathologic classification remain subjects for speculation; secondly, the results of treatment, especially of malignant lesions, have been most discouraging; and finally, the treatment of even the most benign parotid neoplasm is complicated by its intimate relationship with the facial nerve and the threat of disfiguring facial paralysis. Recent contributions by Bailey,[1] Brown,[2] and Kirklin et al[3] have contributed materially to the clarification of the problems and the establishment of sound, definite surgical therapy.

Anatomically, the parotid gland may be described as a dumb-bell shaped organ, with a large, superficial portion lying outside the mandible connected by a slender isthmus to smaller deep portion. McCormack et al[4] have described a natural cleavage plane between these two lobes in which the facial nerve is found. The facial nerve leaves the skull via the stylomastoid foramen, and courses forward to enter the gland at its posterior margin, dividing almost immediately into two trunks which pass around the isthmus. Careful dissections of the nerve[4] have shown some anastomosis between these two trunks around the isthmus in approximately three-quarters of the dissections. This would explain the lower than expected incidence of facial paralysis when individual nerve filaments are divided within the gland. A tongue of gland tissue extending forward along the parotid duct, often termed the accessory parotid gland, is of significance since the removal of a tumor in this portion endangers the duct as well as the superior filaments of the facial nerve.

Pathology

A simple classification of parotid tumors is shown in table 1. The percentages given are those of Brown et al[2] in their series of 149 tumors.

Two-thirds of all parotid tumors fall into the category of benign mixed tumors (fig. 1). Two cell types are found: first, well differentiated epithelial cells with dark nuclei, arranged in sheets or cords; and secondly, connective tissue cells in spindle or stellate arrangement, with a characteristic intercellular mucinous material, often resembling cartilage. While a typical section shows these histologic features, it is not unusual to see tumors in which one element predominates to the exclusion of the other. A section without mucinous connective tissue, and consisting entirely of sheets of epithelial cells is easily mistaken for carcinoma.

There are two current theories as to the origin of parotid gland mixed

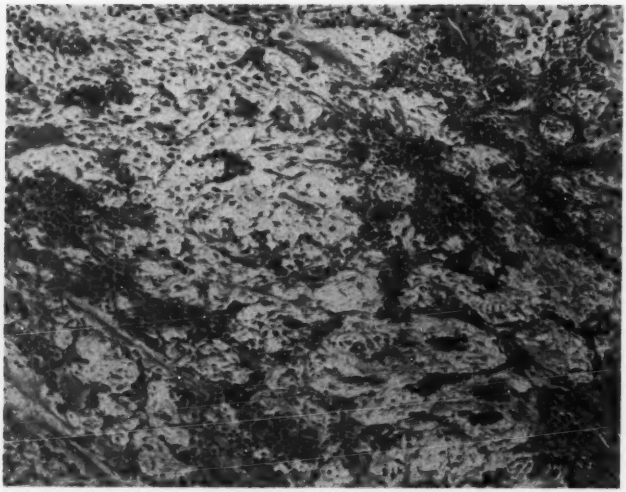


FIG. 1. Typical mixed tumor showing clumps of epithelial cells and mucinous connective tissue (x70).

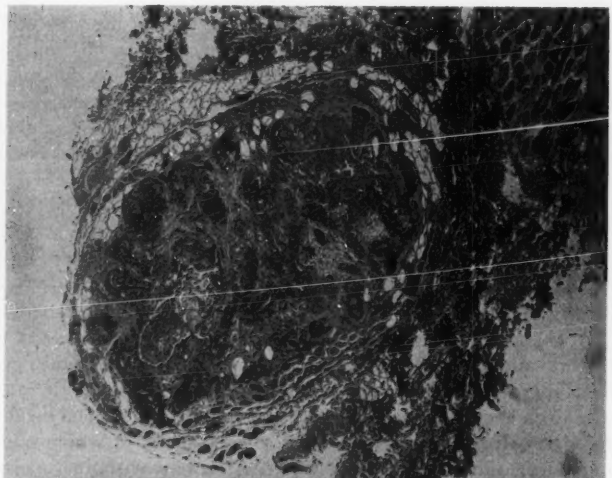


FIG. 2. Malignant mixed tumor showing fat and muscle invasion well outside main tumor (x50).

tumors. Halpert[5] considers them to be anlage tumors, derived from nests of embryonal ectoderm. Willis,[6] in describing microscopic sections in which mixed tumor and normal glandular tissue blend into each other without any visible junction, feels strongly that the tumor is of salivary gland origin. Conclusive evidence on either side is still lacking.

The most common malignancies are the carcinomas, which fall into five groups. The malignant mixed tumor (fig. 2) is characterized by the presence of malignant epithelial cells with mitotic figures superimposed on the mucinous matrix. Invasion through and beyond the fibrous capsule of the tumor is common.

Cylindromatous carcinoma (fig. 3) is composed of sheets or groups of round, regular epithelial cells arranged around hyaline masses. While nerve sheath

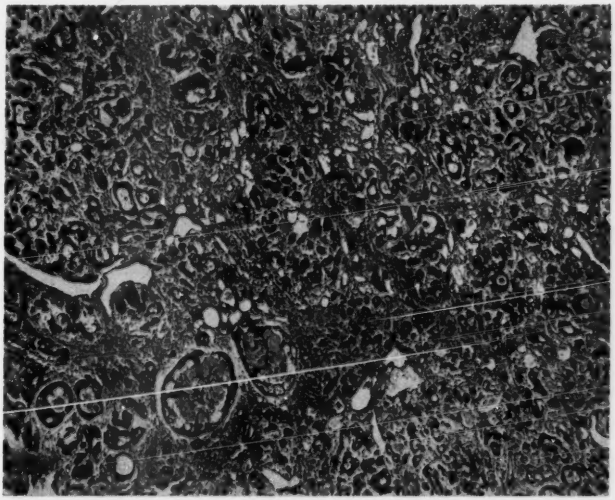


FIG. 3. Cylindromatous carcinoma showing groups of dark staining cells and hyaline masses (x50).

invasion and extension generally accompany this variety of carcinoma, there is often a latent period during which the tumor is relatively benign and curable by simple excision before rapid growth, invasion and metastases occur.

Adenocarcinomas of the parotid (fig. 4) may vary from well differentiated tumors with acini of obvious glandular origin, to anaplastic lesions in which the correct diagnosis is made only with difficulty. Squamous carcinomas are similarly variable, with microscopic pictures ranging from those with typical epithelial pearls to others showing little differentiation. Occasionally, areas of adeno or squamous carcinoma are found within an otherwise unremarkable mixed tumor. Furthermore, adeno and squamous elements may be encountered in the same lesion, with considerable variation in the amounts and degrees of

FIG. 4. Adenocarcinoma with moderate differentiation (x70).

differentiation of the components. A rather unusual member of this group is the "mucoepidermoid carcinoma" described by Stewart, Foote and Becker,[7] consisting of mucus-producing glandular epithelium, groups of cells of squamous type, and areas of pooled mucus (fig. 5).

Less than 5 per cent of all the parotid tumors fall into the category described first by Warthin[8] in 1929, and known as papillary cystadenoma lymphomatosum. The epithelial portion is papillary with prominent cystic spaces. Lymphocytic material is present, scattered irregularly throughout the lesion.

The miscellaneous group of tumors includes typical angiomas of either blood vessel or lymphatic origin, the latter sometimes being found as cystic hygroma with spaces containing typical watery material. Lipomas are rare, but come to operation from time to time with the clinical signs of mixed tumor or papillary cystadenoma lymphomatosum. Benign adenomas are not clinically distinguishable from other benign tumors, and can be diagnosed only after examination of the surgical specimen.

Diagnosis

The patient with a benign mixed tumor usually presents a history of a painless lump in the parotid area, of several months' to many years' duration. It may vary considerably in size, sometimes growing unbelievably large without causing distress sufficient to make the patient seek treatment. Facial paralysis is never present. On examination one finds a firm, freely movable tumor, most commonly in the lower half of the gland.

The diagnosis of carcinoma is obvious if fixation of the mass, or facial

nerve paralysis, is present. Any rapid increase in size of the tumor should make one suspect a malignant lesion. This course is particularly characteristic of cylindromas, whose sudden change in size marks the start of its more malignant phase.

Papillary cystadenoma lymphomatosum (fig. 6) is characteristically soft and diffuse, with vague boundaries, and sometimes bilateral. Unfortunately, other soft tumors may present a similar picture, thus requiring further diagnostic procedures before specific therapy is undertaken.

While the biopsy is one of our soundest surgical fundamentals, its value in the diagnosis of parotid tumors is limited. However, it may be useful in the evaluation of malignant lesions, in order to plan definitive radical surgery prior to operation. In any case, since the entire biopsy field must be excised in continuity with the mass to eliminate the possibility of seeding tumor cells, any biopsy should be planned with care, and the incision placed in such position that it can be easily excised in the course of definitive surgery.

Frozen section is useful, but not necessarily conclusive, even in the hands of a most competent pathologist. If a frozen section is not unquestionably diagnostic, it is far better to utilize the primary excision of the tumor as a biopsy, following it, if necessary, with a second definitive operation when the permanent microscopic sections have been interpreted.

Treatment

The treatment of choice for a tumor diagnosed as benign mixed tumor is early excision. Endotracheal anesthesia is used, care being taken to bring

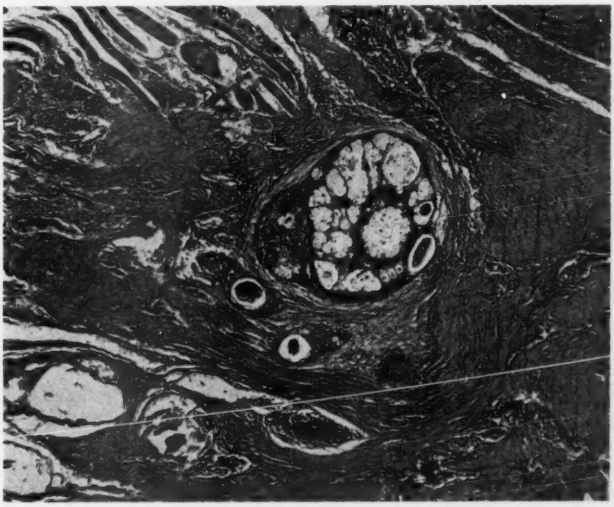


FIG. 5. Mucoepidermoid carcinoma. Note mucus-filled spaces within and outside the area of carcinoma (x50).

the airway out of the side of the mouth away from the operative field. The assistant assigned to watch the face thus has an unobstructed view of twitches resulting from facial nerve stimulation. The incision is designed to be cosmetically minimal yet provide wide exposure of the entire parotid area. It follows the anterior border of the ear, is carried back beneath the lobe, and is then extended down the neck in one of the skin creases. The lower limb of the incision may be lengthened or modified if necessary to open the entire neck for radical lymph node dissection (fig. 7).

It is not our policy to identify the main trunks of the facial nerve prior to approaching the tumor. However, in certain cases it may be useful for orientation to identify some segment of the nerve. This is most simply done by picking up its inframandibular branch at the inferior border of the gland,

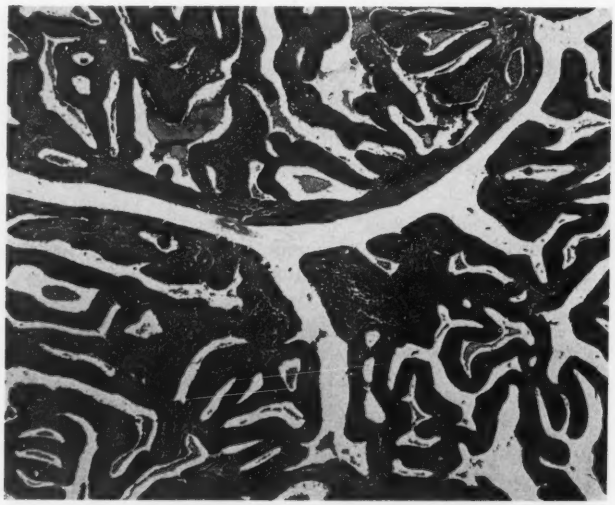


FIG. 6. Papillary cystadenoma lymphomatosum. Warthin's tumor.

where it courses downward a few millimeters anterior to the posterior facial vein. This may be followed backward without difficulty until the main nerve trunk is encountered.[9]

Dissection of the lesion is then carried out by carefully dividing overlying and surrounding soft tissue, while the face is watched for twitches. The attempt is made to leave a thin layer of gland tissue on the tumor; needless to say, this becomes extremely difficult as individual nerve filaments are met lying immediately on the capsule of the tumor. If the lesion is presumed benign, every effort is made to save facial nerve at the risk of leaving behind a few adherent cells. In actual practice, the incidence of recurrence under these circumstances is very low. Dissection is continued around the entire mass, using small scissors

and a fine-tipped hemostat to spread adjacent tissue. Any tissue which must be cut is first stimulated with the hemostat while the face is watched, to avoid dividing an included nerve filament. Bleeding is controlled with fine white silk ties and the operative wound irrigated gently with warm saline solution.

A small rubber band or narrow Penrose drain is placed into the parotid bed and brought out behind the ear. The defect is closed with catgut in the subcutaneous layers, and fine silk in the skin. A voluminous, carefully applied head dressing using mechanic's waste provides adequate pressure to prevent the collection of saliva and blood in the wound.

The drain is removed in 2 or 3 days. Pressure dressing must be continued until the skin flap is well attached to its bed (figs. 8 and 9).

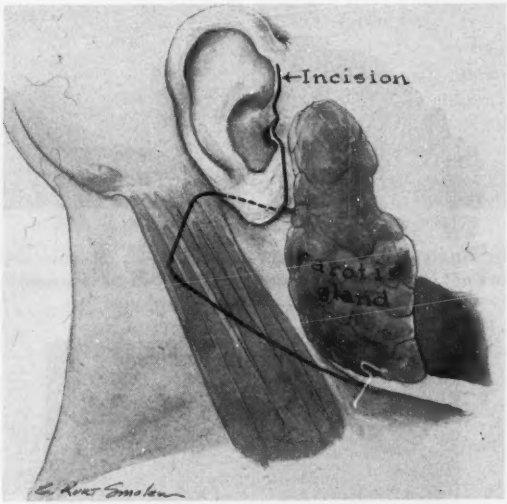
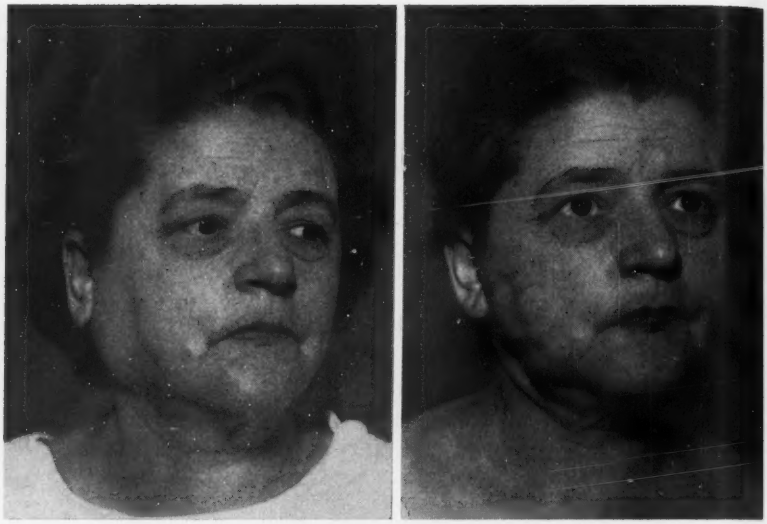


FIG. 7. Incision is designed to provide wide exposure of entire parotid area yet produce minimum cosmetic deformity.

In the case of carcinoma, the facial nerve must usually be sacrificed in the course of radical excision of the tumor. Furthermore, removal in continuity of the regional neck nodes is advisable in most cases. Certain of these malignant lesions are radiosensitive and will respond to massive doses of x-ray or interstitial radium. However, results with radiation are still inconstant and unpredictable, and radical surgery remains our best approach to the problem.

It should be emphasized to the patient who is about to lose his facial nerve that facial paralysis is not an incredible catastrophe. Excellent methods are available to repair the cosmetic deformity, using loops of fascia lata anchored to the temporalis fascia to support the corner of the mouth, upper lip and lower eyelid. It is essential that fear of facial paralysis not be allowed to stand in the way of adequate treatment of cancer.



(a) (b)

FIG. 8. (a) Patient with typical mixed tumor. Tumor is freely movable, low in parotid area, superficial, and has not involved facial nerve. (b) Same patient following excision of tumor.

FIG. 9. Lateral view of patient in figure 8 showing scar 2 weeks after operation.

Papillary cystadenomas, if small, may be easily excised. If large and diffuse, they are best treated with small doses of x-ray, with satisfactory results.

Results

One should expect a recurrence rate of under 5 per cent following careful excision of mixed tumors with preservation of the nerve. In the same cases, the incidence of complete facial paralysis should be zero. Occasional partial palsies may be noted in the immediate postoperative period, as the result of stretching or dividing small nerve filaments. Almost all of these are transient, however, and will disappear within a few months, leaving little or no deformity.

The results following radical surgery for carcinoma are relatively poor. While approximately half of the patients will be alive 5 years after surgery, the percentage of cases free of disease will be considerably less. It is to be hoped that early diagnosis plus more widespread use of the principle of resection in continuity of the primary cancer and its regional lymph nodes will improve the prognosis in these individuals.

Table 1

1. Benign mixed tumor (66.7 per cent)
2. Carcinoma (22.6 per cent)
   a. Malignant mixed tumor
   b. Cylindromatous carcinoma
   c. Adenocarcinoma
   d. Squamous cell carcinoma
   e. Mucoepidermoid carcinoma
3. Papillary cystadenoma lymphomatosum (3.4 per cent)
4. Miscellaneous (7.3 per cent)
   a. Angioma
   b. Lipoma
   c. Adenoma

Summary

1. A pathologic classification of tumors of the parotid gland has been presented.

2. The diagnosis and treatment of these tumors have been discussed and the expected results summarized.

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URETHRAL DIVERTICULUM

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DIVERTICULUM of the female urethra is said to be a rare condition. Thirty-eight women afflicted with this pathologic condition have been seen in this institution since 1932. Engel[1] and Higgins[2] have reported previous cases. Twenty-six of these patients have been observed since the last report in 1945. This increased frequency of diagnosis does not reflect a change in the natural occurrence but instead indicates increased interest in case identification.

A diverticulum represents an outpouching of the ventral wall of the urethra and is, as a general rule, the end result of infection of a periurethral gland. It is usually manifested by a tender palpable swelling along the anterior vaginal wall. There is a direct communication, single or multiple, with the urethra which allows intermittent drainage of the sac contents. With continued adequate drainage the diverticulum may be asymptomatic. Since periurethral glands contain numerous branches and surround the urethra laterally, the diverticulum may be unilocular or multilocular and may even girdle the urethra in a saddle-like fashion.

The following case report is presented to emphasize the clinical features as well as diagnosis and treatment of this condition.

In June 1951, a 45 year old woman came to the Cleveland Clinic complaining of urethral pain and aching, especially with distention of the bladder. She was the mother of two children, her last pregnancy ending uneventfully in 1941. For the past 12 years she had had repeated attacks of dysuria, frequency, and urgency. The patient had noted a swelling in the vagina and experienced dyspareunia when the swelling was prominent and the urinary symptoms prevailed. Throughout the years she had been treated for recurrent cystitis by means of urinary antiseptics and antibiotics with only temporary relief.

Vaginal examination disclosed an exquisitely tender 2.5 cm. mass on the anterior vaginal wall extending almost to the bladder base. Pressure on it caused pus to extrude from the urethral meatus. Panendoscopic examination revealed a small opening in the floor of the middle third of the urethra from which pus could be seen escaping from the diverticulum. A urethrogram showed a large collection of radio-opaque material along the midurethra, outlining a multilocular diverticulum (fig. 1).

The urethrovaginal septum was explored transvaginally and the diverticulum completely excised. The opening into the urethra was closed and allowed to heal for 10 days before removing the catheter. The patient had an uneventful convalescence and has remained symptom free for one year. Vaginal examination now shows no evidence of swelling along the anterior vaginal wall.

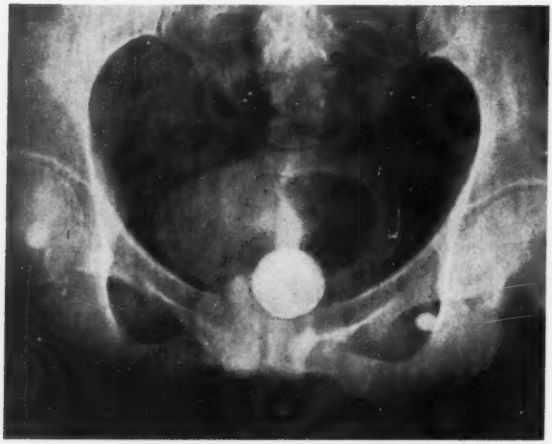

FIG. 1. Large urethral diverticulum. The diverticulum lies under symphysis pubis extending up under the bladder. Symmetrical round shadow is the bag of a Foley catheter filled with radio-opaque material and pulled down snugly against bladder neck.

This patient demonstrates the classical symptoms and findings associated with a urethral diverticulum. She had a long history of lower urinary tract symptoms suggesting recurrent infection. She had noted a swelling within the vagina when the bladder symptoms and dyspareunia were most evident. A cystic mass was palpable along the anterior vaginal wall and pressure on it produced pus at the urethral meatus. The diagnosis was confirmed by the urethroscopic visualization of the openings into the diverticulum and a urethrogram showed its size and extent.

Other symptoms can occur and should be emphasized. Burning at the conclusion of urination and dysuria are usually observed. Some patients experience dribbling of small amounts of urine after voiding due to emptying of the diverticulum. In some instances the diagnosis has to be based only on the history and physical findings because the opening into the diverticulum is closed off and cannot be demonstrated by urethroscopic examination or urethrogram.

In general, if the above diagnostic criteria and methods are kept in mind and employed, there should be little difficulty in making an accurate diagnosis. However, it is important to differentiate a diverticulum from several other conditions of the anterior vaginal wall. Urethrocele (a poor term) means the downward protrusion of the urethra from its attachments beneath the symphysis pubis. This is not a diverticulum but simply a part of the commonly occurring anterior vaginal wall relaxation. Gartner's duct cysts are common, but they are asymptomatic and located lateral to the midline. An adenoma of a urethral gland is uncommon.

Treatment consists of surgical excision of the sac with repair of the urethral defect. Urethral dilatations, vaginal drainage, repeated courses of antibiotics

and other halfway measures have no place in the treatment of urethral diverticula.

Summary

The clinical features of diverticulum of the female urethra are presented. The diagnosis is made on the basis of characteristic symptoms and the presence of a mass along the anterior vaginal wall. The diagnosis is confirmed by urethroscopic examination and urethrography. Treatment consists of surgical excision and repair of the urethra. By calling attention to this abnormality it is hoped that urethral diverticula will be recognized with greater frequency.

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TRANSORBITAL LEUKOTOMY FOR THE PAIN OF MALIGNANT DISEASE

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THE groundwork for all psychosurgery was laid by Egas Moniz.[1] His original monograph, published in 1936, revealed that for some years he had been considering the possibility of interrupting projection fibers from the frontal lobes, as a therapeutic measure in certain psychoses. The first actual operations in this region were conceived and carried out by Moniz and Lima, and from their work have developed all the more recent and elaborate technics.

To Freeman and Watts,[2] however, must go much of the credit for stimulating research along the lines of surgery on the frontal lobes, at least in the United States. The technic of prefrontal lobotomy described by them, together with their encouraging results, gave a tremendous impetus to the study of frontal lobe function from the surgical aspect, and brought psychosurgery to its present status.

Most of the early work was done, of necessity, in the treatment of mental disease, but it became evident to the observers who were following patients postoperatively, that there had been a definite change in the attitude of many patients with severe pain, as shown by the lessened emotional response to their suffering. Consequently, operations on the frontal lobes for the relief of intractable pain became common, and there is now a series of reports[3-10] in the literature concerning the results of these procedures. The technics of operation have varied from the blind section originally described, through various stages to cortical ablation (gyrectomy or topectomy), including cortical undercutting[11] and prefrontal lobotomy under direct vision.[12,13] Selective excision of the postcentral sensory cortex[14] does not really fall into this category, but is one other step in the attempt to treat pain which has failed to respond to less drastic measures.

Division of white fibers in the frontal lobes via the orbital plate was first proposed and carried out by Fiamberti,[15,16] but much of the recent work on this approach to the problem must be credited to Freeman.[17] Transorbital leukotomy is a method of undercutting the cortex of areas 9 and 10 (Brodman) and causes little constitutional upset to the patient. The technic, which has been described elsewhere,[18,19] does not appear to have gained tremendously in popularity. However, it does have a certain advantage which commends it to the surgeon, namely, that it is a relatively minor procedure which can be tolerated by patients too sick to undergo major surgery. For this reason and in spite of the fact that Freeman reported in his first 100 cases that the effects of the operation on pain of long duration were not dramatic, it was felt that it should be given a trial in certain selected patients. The results of frontal lobe operations on patients with intractable pain show considerable variation.

At the outset therefore, it appeared obvious that 100 per cent effectiveness could not be expected from the less mutilating and less complete section of the white fibers by the transorbital route.

The bilateral operation can be completed in 5 to 10 minutes and, although the change of personality is minimal following this procedure, there is often a definite lessening of the mental and emotional depression so often associated with the knowledge of incurable, deteriorating disease.

Another factor which we have felt to be of significance is that following operation it has, in many cases, been easier to control the patient's pain with narcotics even when the operation was not entirely successful in completely relieving the pain. This has often been pointed out in connection with leukotomies of all types, and the improved mental attitude in these patients, whether as a direct result of sectioning of the white fibers or because of reduced narcotic intake, has often been quite pronounced.

We carry out the operation under pentothal anesthesia, and give 5 Gm. of sulfadiazine intravenously after the surgery is completed, in lieu of the penicillin recommended by Freeman.

As with other forms of prefrontal leukotomy, the effect on the patient's pain and attitude toward his discomfort may not be permanent. Consequently, we have confined the use of this procedure to patients in the late stages, usually with only a few months to live, in whom a more major procedure would seem unjustifiable.

An interesting finding following transorbital leukotomy for pain is that, although the immediate effects may not be marked, within 3 to 6 weeks of operation considerable improvement may occur. This came to our attention when one patient on whom the procedure was carried out failed to obtain early relief. We planned to perform a prefrontal topectomy about 5 days after the transorbital leukotomy, but the patient developed an upper respiratory infection and was sent home for 2 weeks. During this period her condition underwent a definite change for the better, and when she returned there was such obvious improvement that further operation was contraindicated. This was not a case of malignancy, but of traumatic neuropathy, and after more than a year the patient has continued to do satisfactorily. Several other patients who have undergone a similar sequence of events have been seen.

We do not carry out transorbital leukotomy in patients with recognized cerebral metastases.

The statistics are as follows:

Number of cases	25
Results—	
Good	11
Fair	5
Poor	7
Fatal	2

A good result is classified as one in which the complaints of pain were noticeably diminished and no sedation was necessary, or in which control of pain by sedation was effected more easily following the operation. Fair results are those in which the patient postoperatively continued to require moderate sedation, but the mental attitude was improved. Poor results are those in which there was no change and heavy sedation was still required.

Of the 2 fatalities, the first was a man in whom the lesion was widespread metastatic teratoma of the testis. It was known preoperatively that, for many weeks, this patient had not been able to breathe when lying down, owing to severe enlargement of the mediastinum by metastases and also metastatic involvement of the lungs. Unfortunately, postoperatively he was placed in the horizontal position; while recovering from the anesthetic he vomited, aspirated vomitus, and died from bronchial and tracheal obstruction. The second death presumably occurred as a result of hemorrhage. The patient failed to recover from the anesthetic and died suddenly.

The only other complication in this group was a temporary cerebrospinal fluid rhinorrhea which occurred in one patient. Of the 25 patients reported, at the last estimate 19 had already died as a result of malignancies.

Discussion

In the treatment of intractable pain, which has failed to respond to more peripheral measures, many operations on the frontal lobes have been performed. The results of all these procedures appear comparable, and in our experience, transorbital leukotomy is the least traumatic, the one likely to cause the least personality change, and the one most justifiable in the terminal stages of malignant disease. By this operation, as with the other more radical procedures, apprehension may be alleviated, narcotic intake diminished, and the final weeks or months of a progressive downhill course be made more tolerable for both patient and attendants.

Summary

1. Twenty-five patients in states of terminal malignancies were subjected to transorbital leukotomy.

2. Sixteen patients were improved; 2 died and 7 remained unchanged.

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17. Freeman, W.: Transorbital leucotomy. Lancet **2**:371 (Sept. 4) 1948.
18. Idem: Treatment of intractable pain by transorbital lobotomy. Paris med. **40**:169 (March 25) 1950.
19. Williams, J. M. and Freeman, W.: Transorbital lobotomy in relief of intractable pain. Arch. Surg. **63**:203 (Aug.) 1951.

Certificate Ceremony and Prize Awards

On June 17 certificates were awarded to 16 Fellows who had completed their formal training in the following fields: Surgery: D. E. King, J. S. McKee, Jr., M. R. Richards, Jr. (in absentia), J. E. Scheid, C. Y. Thomas, III (in absentia). Orthopedics: J. W. Hutchison, W. R. Kohlheim. Ophthalmology: George Lockhart, III. Neurology: J. G. Klotz, Alexander Ling. Internal Medicine: K. D. Arn, R. I. Cottle, Jr., R. C. Netherton, M. B. Shaw, N. W. Zaworski, R. E. Zwickel.

The 1952 Lower Fellowship Thesis Prize was presented to Dr. William John Krech for his article entitled *Meniere's Disease: Treatment by Labyrinthotomy and Electrocoagulation.* Dr. Krech, a former Fellow in Otolaryngology, is now located in Columbus, Ohio. The prize was established in 1935 by Dr. William E. Lower.

At the same ceremony the Assistant Staff Prize for outstanding publication in the literature was awarded to Dr. Penn G. Skillern for *Cases of Graves' Disease Resistant to Radioactive Iodine.* This paper was published in the Transactions of the American Goiter Society.

RECENT PUBLICATIONS BY MEMBERS OF THE STAFF

Bates, G. C. and Brown, C. H.: Incidence of gall bladder disease in chronic hemolytic anemia (spherocytosis). Gastroenterology **21**:104 (May) 1952.

Beattie, J. and Corcoran, A. C.: Renal clearances of polysaccharide; observations on glomerular porosity and on relation of this function to proteinuria in renal disease. J. Clin. Investigation **31**:445 (May) 1952.

Bennett, H. S. and Collins, E. N.: Oil granuloma of peritoneum. Gastroenterology **20**:485 (March) 1952.

Brown, C. H. and Michels, A. G.: Intussusception in adults. Surgery **31**:538 (April) 1952.

Brown, H. B. and Page, I. H.: Mechanism of iodide action on cholesterol and sterol digitonides in hyper- and hypothyroidism. Circulation **5**:647 (May) 1952.

Collier, F. C., Reich, A. and King, J. W.: Importance of sheep cell suspension in complement fixation reaction; description of accurate technic for preparation of a uniform cellular suspension. J. Lab. and Clin. Med. **39**:778 (May) 1952.

Idem: Role of urea-splitting bacteria in urolithiasis; description of simplified urea-agar medium for their detection. Connecticut State M.J. **16**:257 (April) 1952.

Crile, G., Jr. and Turnbull, R. B., Jr.: Changing trends in surgery of colon. J. Iowa State M. Soc. **38**:12 (Jan.) 1952.

Ernstene, A. C.: Diagnosis and treatment of coronary heart disease. Pennsylvania M.J. **55**:313 (April) 1952.

Fisher, E. R., Neering, J. C., Hazard, J. B. and Hays, R. A.: Effects of radioactive colloidal gold (AU_{198}) on guinea pigs. Am. J. M. Sc. **223**:502 (May) 1952.

Fisher, E. R. and Brown, C. H.: Linitis plastica carcinoma of stomach with extensive metastases simulating colonic lesion. Gastroenterology **20**:503 (March) 1952.

Fisher, E. R. and Turnbull, R. B., Jr.: Malignant polyps of rectum and sigmoid; therapy based upon pathologic considerations. Surg., Gynec. and Obst. **94**:619 (May) 1952.

Gardner, W. J., Wise, R. E., Hughes, C. R., O'Connell, F. B., Jr. and Weiford, E. C.: X-ray visualization of intervertebral disk; with consideration of morbidity of disk puncture. Arch. Surg. **64**:355 (March) 1952.

Johnston, C. R. K.: Chemical agents unsuccessfully employed as substitutes for ACTH and cortisone. Ann. Allergy **10**:197 (March-April) 1952.

Kennedy, R. J. and Lockhart, G., III.: Paresis of abducens nerve following spinal anesthesia. Anesthesiology **13**:189 (March) 1952.

Kennedy, R. J. and McGannon, W. J.: Stellate-ganglion block in retinitis pigmentosa. Arch. Ophth. **47**:287 (March) 1952.

Masson, G. M. C., Corcoran, A. C. and Page, I. H.: Renal and vascular lesions elicited by "renin" in rats with desoxycorticosterone hypertension. Arch. Path. **53**:217 (March) 1952.

Page, I. H.: Humoral and vasomotor controls of blood vessels. Bull. New York Acad. Med. **28**:131 (March) 1952.

Scheid, J. E. and Crile, G., Jr.: Metastasis of carcinoma of stomach to submucosa of rectum. Am. J. Surg. **83**:827 (June) 1952.

Schneider, R. W., Van Ommen, R. A. and Hoerr, S. O.: Hereditary occurrence of testes and absence of sexual hair in amenorrheic women; type of pseudohermaphroditism. J. Clin. Endocrinol. **12**:423 (April) 1952.

Thomas, C. Y., Jr., Fisher, E. R., Turnbull, R. B., Jr. and Krieger, J. S.: Arrhenoblastoma of ovary; report of three cases. Ann. Surg. **135**:543 (April) 1952.

THE FRANK E. BUNTS INSTITUTE

Announces the following Postgraduate Continuation Courses for October 15 and 16, and for October 29 and 30, 1952.

SURGERY OF THE ABDOMEN

Tentative Program

Wednesday, October 15, 1952

8:00-9:00 a.m.	Registration	
	Morning Session	R. S. DINSMORE, M.D., Presiding
9:00 a.m.	Opening Remarks	R. S. DINSMORE, M.D.
9:05 a.m.	Appraisal of the Cardiac Risk in Abdominal Surgery	A. C. ERNSTENE, M.D.
9:25 a.m.	Anesthesia in Surgery of the Abdomen	D. E. HALE, M.D.
9:45 a.m.	The Abdominal Incision	S. O. HOERR, M.D.
10:00 a.m.	Transfusions in Surgery of the Abdomen	R. B. TURNBULL, JR., M.D.
10:20 a.m.	Intermission	
10:30 a.m.	The Role of Esophagoscopy in Surgery of the Abdomen	H. E. Harris, M.D.
10:50 a.m.	Abdominal Pain of Genitourinary Origin	C. C. HIGGINS, M.D.
11:10 a.m.	The Ovaries and the General Surgeon	J. S. KRIEGER, M.D.
11:30 a.m.	The Surgical Treatment of Hernia	A. H. ROBNETT, M.D.
11:50 a.m.	Surgery of the Adrenal Glands	E. F. POUTASSE, M.D.
12:10 p.m.	Luncheon—Courtesy Bunts Institute	
	Afternoon Session	GEORGE CRILE, JR., M.D., Presiding
2:00 p.m.	Treatment of Duodenal Ulcer	GEORGE CRILE, JR., M.D.
2:20 p.m.	The Problem of Gastric Ulcer	S. O. HOERR, M.D.
2:40 p.m.	Cancer of the Stomach	C. H. BROWN, M.D.
3:00 p.m.	Gastroscopy and Gastric Diagnosis	H. R. ROSSMILLER, M.D.
3:20 p.m.	Lesions at the Diaphragm	D. B. EFFLER, M.D.
3:50 p.m.	Panel—Postoperative Complications	R. S. DINSMORE, M.D. (Moderator) J. E. Dunphy, M.D. (Guest), D. M. Glover, M.D. (Guest) George Crile, Jr., M.D., S. O. Hoerr, M.D.
6:00 p.m.	Dinner—Courtesy Bunts Institute	
7:30 p.m.	Evening Lecture—Acute Postoperative Pancreatitis	J. E. DUNPHY, M.D. (Guest)

Thursday, October 16, 1952

	Morning Session	S. O. HOERR, M.D., Presiding
9:00 a.m.	Acute Intestinal Obstruction, Large Bowel	R. B. TURNBULL, Jr., M.D.
9:20 a.m.	Acute Intestinal Obstruction, Small Bowel	A. H. ROBNETT, M.D.
9:40 a.m.	The Pathologist Looks at Rectal Polyps	J. B. HAZARD, M.D.
10:00 a.m.	Management of Cancer of the Rectum	R. B. TURNBULL, JR., M.D.
10:20 a.m.	Intermission	
10:30 a.m.	Management of Advanced or Recurrent Cancer of the Colon	J. E. DUNPHY, M.D. (Guest)
10:50 a.m.	Ulcerative Colitis	E. N. COLLINS, M.D.

11:10 a.m. . . . Ileostomy with Skin Graft (moving picture) . ROBIN ANDERSON, M.D.
11:30 a.m. . . . Panel—Surgery of the Colon . GEORGE CRILE, JR., M.D. (Moderator)
J. E. Dunphy, M.D. (Guest), R. B. Turnbull, Jr., M.D.
E. N. Collins, M.D.
12:15 p.m. . . . Luncheon

Afternoon Session . . . . . R. B. TURNBULL, JR., M.D., Presiding
2:00 p.m. . . . Indications for Cholecystectomy . . . . . R. S. DINSMORE, M.D.
2:20 p.m. . . . Management of Acute Cholecystitis . . . J. E. DUNPHY, M.D. (Guest)
2:50 p.m. . . . Chronic Recurrent Pancreatitis . . . . . GEORGE CRILE, JR., M.D.
3:10 p.m. . . . Operative Cholangiography (moving picture) . . . S. O. HOERR, M.D.
3:30 p.m. . . . Technical Problems in Splenectomy . . . . . R. S. DINSMORE, M.D.
3:50-5:00 p.m. . Panel—The X-ray in Diagnosis of
Abdominal Surgical Diseases . . . S. O. HOERR, M.D. (Moderator)
J. E. Dunphy, M.D. (Guest)
C. R. Hughes, M.D., George Crile, Jr., M.D.
R. B. Turnbull, Jr., M.D., E. N. Collins, M.D.

MANAGEMENT OF COMMON DERMATOLOGIC PROBLEMS

Tentative Program

Wednesday, October 29, 1952

8:30-9:00 a.m. . Registration

Morning Session—E. W. Netherton, M.D., Presiding
9:00 a.m. . . . Opening Remarks . . . . . F. A. LEFEVRE, M.D.
9:10 a.m. . . . Atopic and Contact Dermatitis . . . . . E. W. NETHERTON, M.D.
9:30 a.m. . . . Seborrheic Dermatitis . . . . . G. H. CURTIS, M.D.
9:50 a.m. . . . Fundamentals of Dermatologic Therapy . . E. W. NETHERTON, M.D.
10:10 a.m. . . . Surgical Procedures and their Indications in
Varicose Ulcers and Stasis Dermatitis . . . . A. H. ROBNETT, M.D.
10:30 a.m. . . . Intermission
10:45 a.m. . . . Present Day Concepts of the Management
of Syphilis . . . . . A. C. CURTIS, M.D. (Guest)
11:30 a.m. . . . Use and Abuse of Cortisone and ACTH in
Management of Common Dermatoses . . . . J. R. HASERICK, M.D.
11:50 a.m. . . . Treatment of Hemangiomas . . . . . U. V. PORTMANN, M.D.
12:10 p.m. . . . Surgical Management of Pruritus Ani
et Vulvae . . . . . R. B. TURNBULL, JR., M.D.
12:30 p.m. . . . Luncheon—Courtesy of the Bunts Institute

Afternoon Session
1:45 p.m. . . . Special Lecture—Subject to be announced A. C. CURTIS, M.D. (Guest)
2:30 p.m. . . . Common Superficial Fungus Infections . . . . . G. H. CURTIS, M.D.
2:50 p.m. . . . Cutaneous Manifestations of Peripheral
Vascular Disease . . . . . V. G. deWOLFE, M.D.
3:10 p.m. . . . Moles, Keratoses, and Verrucae . . . . . J. R. HASERICK, M.D.
3:30 p.m. . . . Urticaria . . . . . C. R. K. JOHNSTON, M.D.
3:50 p.m. . . . Intermission
4:00 p.m. . . . Stress Dermatoses . . . . . G. H. CURTIS, M.D.
4:20 p.m. . . . Lupus Erythematosus . . . . . J. R. HASERICK, M.D.
4:40-5:00 p.m. . Questions and Answers

Thursday, October 30, 1952

Morning Session

9:00 a.m. . . . Papulosquamous Dermatoses: Psoriasis, Lichen Planus, Pityriasis Rosea . . . . . E. W. NETHERTON, M.D.

9:20 a.m. . . . Cutaneous Syphilis . . . . . G. H. CURTIS, M.D.

9:40 a.m. . . . Role of Plastic Surgery in Dermatologic Therapy . . . . . ROBIN ANDERSON, M.D.

10:10 a.m. . . . Surgical Lesions of Mouth and Pharynx . . . . . H. E. HARRIS, M.D.

10:30 a.m. . . . Intermission

10:45 a.m. . . . Cutaneous Manifestations of Common Virus Diseases . . . . . J. R. HASERICK, M.D.

11:00 a.m. . . . Acne Vulgaris and Rosacea . . . . . G. H. CURTIS, M.D.

11:20 a.m.-12:00 p.m. Noon Panel—Antibiotics . E. W. NETHERTON, M.D. (Moderator)
G. H. Curtis, M.D., J. R. Haserick, M.D.
C. R. K. Johnston, M.D., V. G. deWolfe, M.D.

Guest Speakers:

John E. Dunphy, M.D.—Associate Professor of Surgery, Harvard Medical School, Boston, Mass.

Donald M. Glover, M.D.—Director of Surgery, St. Luke's Hospital, and Associate Clinical Professor, Western Reserve University School of Medicine, Cleveland, Ohio.

Arthur C. Curtis, M.D.—Professor of Dermatology and Syphilology, University of Michigan Medical School, Ann Arbor, Mich.

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THE FRANK E. BUNTS INSTITUTE
Cleveland Clinic
East 93rd Street and Euclid Avenue
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Please register me for the course on "Management of Common Dermatologic Problems" to be given October 29 and 30, 1952. (Registration Fee is $15.00, except for interns and residents, and members of the Armed Forces in uniform, who will be admitted free.)

I am enclosing check for $5.00 and the remainder will be paid on registration, October 29. Checks should be made payable to the Frank E. Bunts Institute.

Name .....

Address .....

Medical School and
Date of Graduation .....

This course is open only to graduates of approved medical schools.

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Cleveland Clinic
East 93rd Street and Euclid Avenue
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